VOLUME I—NUMBER 3 MARCH 1950

UNITED STATES ARMED FORCES

MEDICAL JOURNAL

*Published monthly by the Department of Defense*

JOSEPH L. SCHWARTZ, *Captain (MC), U. S. N.*, Editor in Chief
WAYNE G. BRANDSTADT, *Lieutenant Colonel MC, U. S. A.*, Associate Editor
ALFRED A. GENTILCORE, *U. S. A. F. (MSC)*, Associate Editor

THE PRINTING OF THIS PUBLICATION HAS BEEN APPROVED BY THE DIRECTOR OF THE BUREAU OF THE BUDGET, OCTOBER 19, 1949.

# Malignancies of the Colon[1]

## A Discussion of Some Problems

ISIDOR S. RAVDIN, *B. S., M. D.*[2]

IN THE field of intestinal surgery even competent surgeons are not agreed on the solution of a number of problems among which are the following: what constitutes operability; should a radical operation be performed in the presence of major metastasis; what are the advantages of a one-stage over a two-stage operation; aseptic versus open anastomosis; or various modifications of the Mikulicz operation versus end-to-end or end-to-side anastomosis. The well-known caution of our profession has led us frequently to continue concepts and procedures which in the light of modern evidence are outmoded, or, if one searches carefully for the truth, not in the best interests of the patient. The answer to many of these problems is not to be found in statistical analysis alone, for operability varies with the skill and boldness of the surgeon, method often varies with training and expe-

[1] Presented at the Monthly Medical Meeting, Army Medical Center, Washington, D. C., on 15 December 1949.

[2] From the Surgical Clinic of the Hospital of the University of Pennsylvania and the Harrison Department of Surgical Research, Schools of Medicine, University of Pennsylvania, Philadelphia.

rience, and both may depend on a consideration of the immediate mortality and not on the more remote factor of end results. The operative treatment of lesions of the colon presents the surgeon with a multitude of difficult problems in diagnosis, in judgment, in pre- and post-operative care, and in technique.

The diagnosis of large bowel malignancy should not be difficult and yet more than 50 percent of our patients come to us with metastasis to abdominal lymph nodes or to the liver. More than 50 percent of the cancers of the large bowel including the rectum are within reach of the finger, the proctoscope, or the sigmoidoscope. Medical students spend endless time looking into the nose, mouth, pharynx, and the external auditory meatus, as part of a so-called complete examination of the patient, while the examination of the rectum is neglected. Few of our medical schools insist on the students learning the technique of proctoscopic and sigmoidoscopic examination. A change in bowel habits, a grave and unexplained anemia, continuing colic, the presence of blood in the stools, all call for a careful examination of the abdomen, proctoscopic and sigmoidoscopic examination, and a careful radiographic study of the large bowel above the rectosigmoid with barium enema regardless of the age of the patient.

A cancer of the cecum, ascending colon, and often of the hepatic flexure is apt to be a moderately large tumor extending into the lumen of the bowel. Only rarely is it an annular scirrhous lesion. This portion of the bowel has a larger diameter, and the fecal content is liquid or quite soft and, therefore, acute intestinal obstruction is rare. Anemia, on the other hand, is frequent and may be so severe as to simulate a primary anemia. It results in part from blood loss from the surface of the tumor and perhaps in part from blood destruction, but the cause of the latter is not now known.

In the transverse colon the fecal stream becomes firmer, the lumen of the bowel is smaller and the tumor tends to become annular. Constipation and colic will be found in a high percent of the patients with malignant lesions in this area.

In the splenic flexure one of the most common features of cancer is the frequency with which perforation and a left subphrenic abscess occur. As one follows the lesions to the rectosigmoid, they become even more annular and scirrhous. The fecal stream becomes more solid and obstruction occurs with increasing frequency. Even in the absence of acute obstruction one often finds considerable distention of the bowel proximal to the lesion, and the presenting symptoms are so frequently associated with massive cecal distention as to suggest a lesion in the right lower abdominal quadrant.

In the rectum one finds a variety of lesions, some sessile, some excavating, others polypoid; the presenting symptoms may vary with

the type of lesion that is present. There are, of course, variations from these pathologic characteristics, such as when cancer is engrafted on a single polyp of the colon or occurs in one or more areas of polyposis.

In the past year and a half we have operated on 14 patients with more than 1 primary malignant lesion of the large bowel. In every instance these lesions have been engrafted on preexisting adenomatous polyps.

At least two types of colonic polyposis occur; one is associated with prolonged infection of the colon, and the other is a congenital lesion. The first is pathologically and significantly different from the second. The polyps, which have their origin in a bowel that is the site of chronic colitis or recurring dysentery, are myxomatous in character and are like the polyps so frequently seen in association with long-standing maxillary sinusitis.

The adenomatous polyp is a true tumor. It is frequently seen in several members of the same family. It may be found in children if careful studies are made. I have within the past year, removed a large adenomatous polyp from a child 3 years of age. The adenomatous polyp must be looked on as a potentially serious lesion for 50 percent or more of patients with generalized adenomatous polyposis will have malignant lesions before the fortieth year and nearly 100 percent by the age of 65.

If there is a hereditary precancerous lesion it is congenital adenomatous polyposis. This lesion accounts for nearly 100 percent of the instances of familial cancer of the large bowel. Every officer and enlisted man inducted into the Armed Forces should be questioned as to the cause of death of members of his family. If colonic cancer has been a cause of death, a careful study should be made for the presence of adenomatous polyps.

Cancer occurs much less frequently in the large bowel that is the site of the polyposis associated with chronic infection. Dennis has reported the presence of cancer in 12 percent of the colons he has resected for chronic ulcerative colitis. The cancer in these instances is not, however, engrafted on the polyposis.

It has been stated that resection of the colon down to the sigmoid, and fulguration of the remaining polyps in the sigmoid and rectum, if they are not too numerous, followed finally by an ileosigmoidostomy is a satisfactory procedure for adenomatous polyposis. It may well be when the polyps in the rectum and sigmoid are few in number and when the patient can be kept under constant surveillance. The following case report illustrates what can happen when too conservative measures are used, and emphasizes the responsibility of the

surgeon to perform the most radical operation possible if any operation is to be performed.

### CASE REPORT

The patient, a 23-year-old woman, had widespread colonic polyposis of the adenomatous type. One grandfather, one uncle, and her father had died of intestinal malignancy, presumably engrafted on colonic polyposis. Her intense desire to save her rectum had led an able surgeon to perform a colectomy to just above the rectosigmoid and later to perform an ileoproctostomy. The polyps remaining in her rectum were fulgurized. There was no follow-up after the first year when she was pronounced "out of danger," and yet 2 years later I saw her with widespread rectal cancer and hepatic metastasis. She died about 13 months later.

I have seen other instances of this type, which fortify my conviction that only a complete operation should be performed and that no operation is complete without resection of the entire polypoid area unless the patient can be kept under frequent observation.

Only a small percent of the total number of patients with cancer of the colon treated in any clinic are admitted with acute obstruction. These require an emergency operation. The nonobstructed patients are greatly benefited by a period of from 7 to 10 days of careful preoperative preparation. This is true whether the lesion is in the right or left half of the colon. It should be dispensed with only when the contemplated operation is to be a proximal colostomy.

The majority of these patients are malnourished in varying degrees. Many more are anemic. Loss of sleep from pain, a debilitating diarrhea, and abdominal distention, together with anemia and hypoproteinemia, combine to make the patient a poor operative risk unless a carefully designed preoperative program is initiated which will lead to improvement in nutrition and anemia, to better circulation, and to a reduction in the bacterial count of the intestinal contents. Such a program will permit more radical operations to be performed in one stage, with a lessened incidence of postoperative peritonitis, serious wound infections, and wound dehiscence. It will tend to reduce the incidence of exaggerated edema at the sites of resection and anastomosis. Edema at such a site follows all such operations, but it is intensified when the biologic mechanism for returning fluids to blood vessels is disturbed. The control of this is of real importance when resection and immediate suture is contemplated, for the brawny edema that follows trauma in the hypoproteinemic patient may, for some days, prevent the normal functioning of an otherwise perfect end-to-end anastomosis and may result in leakage at the site of suture from faulty wound healing.

In the absence of definite obstruction the patient should be fed by mouth while the bowel is being prepared and the condition of the patient is being improved. Deficiencies in the nutritional state of the

patient can be corrected by the administration of a high carbohydrate, high protein, diet containing a moderate amount of fat. There is no need to eliminate fat from the diet of these patients. If 25 percent or more of the total calories are derived from protein, fatty infiltration of the liver does not occur. The fat will provide twice the number of calories, gram for gram, as will carbohydrate or protein. The diet should, we believe, always be a low-residue diet in order to facilitate the ultimate aim of a clean bowel. It should provide 3,000 to 3,500 calories per day.

The vitamin intake should be ample not only for the usual vitamin requirements but also for complete physiologic requirements since vitamin synthesis from intestinal bacteria may be seriously impeded once oral chemotherapy or antibiotic therapy is begun. We must be more alert to the serious nutritional changes that may take place when effective intestinal bacterial growth is greatly retarded. Not only may prothrombin synthesis be effected, but amino acid utilization becomes impaired when, as the result of intestinal bacterial decimation, riboflavin and other vitamins are no longer synthesized in adequate amounts. The amounts of the various vitamins that we consider necessary for oral ingestion are greatly supplemented by intestinal bacterial synthesis to meet our physiologic requirements, and the amounts synthesized within the bowel may be in excess of those ordinarily ingested.

Dependence for the correction of the anemia should not be placed on diet and iron but on multiple transfusions. When anemia and a protein deficiency coexist, the protein ingested above that required for energy will go first to the synthesis of hemoglobin and not to replace the depleted stores of body and plasma protein. In the severe anemias so frequently observed in patients with cancer of the cecum and ascending colon, it may not be possible to correct the anemia completely prior to resection. In such patients rapid restoration of the necessary blood following operation is a very useful procedure, but the restoration of plasma volume and blood mass by transfusions prior to operation make them better operative risks.

Chemotherapy is begun as soon as the patient is admitted to the hospital and the diagnosis established. Succinylsulfathiazole is as effective an agent as is now available for the initial therapy. Our patients receive 4 to 6 gm. of this drug every 6 hours by mouth, for 5 to 7 days. In the last 3 days of preoperative preparation, 0.25 gm. of streptomycin is given orally every 6 hours. With this combination effective bacterial control is obtained and bacterial resistance to these agents is not frequently encountered. There is no doubt in our minds that streptomycin has a wider antibacterial spectrum than does any other agent now available. Its use after 4 or 5 days may be followed by a specific resistance of certain organisms and it is for this reason

that the combined therapy has received wider use. The use of effective bacteriostatic and bacteriocidal agents by mouth before operation, the parenteral administration of penicillin and streptomycin in the postoperative period, together with an improved nutritional state that will permit the mobilization of a normal biologic defense mechanism, are all adjuvants to sound surgery. The result is a distinct reduction in morbidity and mortality in patients requiring colonic resection.

Of even greater importance is the fact that we have been able to change from modifications of the Mikulicz procedure to the more desirable resection and direct anastomosis. No attempt is made to perform an entirely aseptic anastomosis. We agree with Karl Meyer that if the patient is properly prepared, open anastomosis is a safe procedure.

In 1940, William Osler Abbott and I reported on the use of the Miller-Abbott tube as a means of facilitating a one-stage resection and anastomosis of the right colon. The use of this method has since been favorably reported on by Whipple and by Newton and Blodgett. When the tube has been successfully passed into the lower ileum or the right colon, the bowel is relieved of distention and normal peristalsis is reestablished; although a completely clean bowel is not obtained, the fecal content is minimal.

The tube is passed 48 hours before the contemplated operation, and its position is checked at intervals by fluoroscopic study. Feeding and chemotherapy are continued during this period. If a right colectomy is performed, the tube is kept in the ileum about 18 inches from the site of the end-to-side anastomosis. If resection of the distal colon is carried out, the tube is left in the proximal colon if it has passed the ileocecal valve. The Miller-Abbott tube remains in the bowel until the patient's intestinal functions have returned to normal, usually the fourth to sixth day after operation. Once peristalsis is reestablished and gas and feces are passed by colostomy or by rectum, it is safe to remove it.

It must be emphasized that the use of the Miller-Abbott tube does not obviate the necessity of a colostomy in cancer of the bowel associated with acute or even with severe chronic obstruction, but even in many of these patients it reduces ileus, adds greatly to the comfort of the patient and to the effectiveness of the colostomy.

In Whipple's series the mortality of resections of the large bowel prior to the use of the tube was 18.2 percent and subsequent to it 2.8 percent. In Newton and Blodgett's series and in our own the use of the tube and chemotherapy were begun at about the same time. The former authors report a reduction in serious infections from 43 to 6 percent when only patients having resection and anastomosis were compared. There was a gross reduction in complications of from

58 to 25 percent and a general reduction in mortality from 22 to 3 percent. Our data are in accord with those just reported, the mortality having been reduced from 18.4 percent prior to chemotherapy and suction to 3.4 percent since.

There has been much difference of opinion as to what operation should be performed in the presence of acute obstruction—a cecostomy or a colostomy proximal to the point of obstruction? In our opinion there is no choice. It is a fortunate circumstance that acute obstruction occurs, nearly always, distal to the hepatic flexure, for this permits the performance of a colostomy, which will later assure a safer radical operation.

A cecostomy provides neither an empty nor a clean bowel. It is necessary to divert the fecal stream completely to obtain this. Fecal soiling can rarely be prevented at the time of radical resection if dependence has been placed on a cecostomy. Fallis has recently stated:

> The uncertainties of appendicostomy and the hazards of cecostomy can be circumvented by utilizing the transverse colon as the site for decompression. Moreover, colostomy in the transverse colon produces not only decompression, but defunctionalization as well. The transverse colon is well suited for the performance of colostomy since it is the most mobile, the narrowest, and the least distensible portion of the colon. Furthermore, if the right half is used, the colostomy is then located well away from any incisions planned for operative procedure on the left colon. Left subcostal, left rectus, transverse at the umbilicus, and the Cherney modification of the Pfannenstiel incisions can all be planned and executed through uncontaminated areas.

I find myself in complete agreement with this statement.

There is no unanimity regarding the general types of operations which should be used in attempting radical resection of the colon for cancer. The truth is that no one operation should be used in all instances and that the surgeon who uses only a single procedure fails to provide the maximum benefit to his patients. There can be no doubt that indications exist for modifications of the Mikulicz operation, but they are becoming rarer with each succeeding year. There has been, however, a sufficient critical experience for one to venture certain generalizations in regard to operative procedures.

The operation should be one which will provide for radical removal of the diseased bowel and maximal excision of the lymph nodes which act as receptacles for the lymphatic vessels from the involved area. No operation can be considered too big and Lahey's dictum of "a large operation for a small tumor" must constantly be followed. The one-stage operation should be performed in every instance when this is possible and when the patient is adequately protected. Two operations subject the patient to the double risk of anesthesia and operation and to prolongation of convalescence. All too frequently trauma at the first operation encourages metastasis and even if it does not, a tumor

not resectable at the first operation in the absence of obstruction is not resectable later.

Regional lymph node involvement is not a positive indication of metastasis, for, in the ulcerating lesions, secondary infection is common, and many of the nodes on microscopic study show only the hyperplasia of chronic infection. Nor should the size of the lymph nodes deter the surgeon from proceeding with a radical resection. Large lymph nodes often merely show inflammation while small ones may be completely filled with neoplastic cells. The resection of small portions of the bowel and even smaller areas of the mesentery will be followed by a higher incidence of early and late metastases than if wide areas of the bowel and mesentery are resected at the first operation. The ideal condition for resection is a clean and empty bowel. This cannot always be attained, and certain advances in preoperative preparation may make it less necessary. It is an ideal to strive for regardless of the type of operation to be performed, and the more nearly it is attained the less risk the operation will entail.

The blood supply of the large bowel is not as free as that of the small bowel, and it is more easily impaired by poorly placed sutures, by too many sutures, and by lack of attention to the vessels supplying a particular segment of the bowel. The wall of the colon is thinner than that of the small bowel except in the rectum, and large portions of it are not so completely surrounded by peritoneum. Complete serosal approximation is, therefore, not always attainable.

Resectability should not be determined with one eye on the immediate mortality. A high resection rate will be followed by a larger number of 5-year cures, but a high total early mortality. The higher the resection rate the larger will be the number of 5-year cures regardless of the immediate mortality. The latter is of no special significance for all patients will be dead in 5 years if nothing is done. If wide areas of the bowel containing the tumor, the contiguous mesentery, and the adjacent lymph node-bearing area can be resected, radical operation should always be attempted.

One must not be deterred by the magnitude of the operation or at times even by distant metastasis. A patient with a resectable lesion of the lower bowel with metastasis to the liver will live his final period of life in greater comfort than he will if the lesion is left behind to involve the bladder and the regional nerve supply. Even if the patient eventually dies from metastasis, so do many of our patients in whom no distant spread is found at the primary operation. A more enlightened viewpoint on resection of parenchymal metastasis is now opening a new era in the surgery of malignant disease.

In lesions of the cecum, ascending colon, and hepatic flexure a one-stage operation is performed, resecting the terminal ileum and right colon. Intestinal continuity is restored by an end-to-side or end-to-

end anastomosis—ileum to transverse colon. In lesions of the transverse colon resection and end-to-end anastomosis is to be preferred to any modification of the Mikulicz operation. In certain of these lesions a preliminary cecostomy may be entirely proper. In lesions of the splenic flexure and descending colon direct anastomosis again is preferable as it is in the proximal sigmoid. In the aged and in those with extensive collateral disease a Rankin obstruction resection is often indicated, but even in these patients, as our experience has become greater, we have come to prefer end-to-end anastomosis.

In the low sigmoid, rectosigmoid, and lesions of the ampulla of the rectum, resection and end-to-end suture have been advocated by Babcock and Bacon, Wangensteen, and Waugh and Custer. The studies by Glover and Waugh on the spread of cancer from this portion of the bowel show that retrograde metastasis to the lower perirectal lymph nodes rarely occurs. Their investigations would seem to provide the necessary evidence that the operation of proctosigmoidectomy is at times justifiable. I do not believe, however, that it is possible to perform the wide dissection of the pelvic floor and perirectal tissues when proctosigmoidectomy is contemplated, which is possible when a complete abdominoperineal operation in the Miles tradition is carried out.

If in each instance the surgeon considers that his final responsibility to the patient and to society is the highest possible percent of 5-year cures, he will think less and less of catering to the immediate whims of the distraught patient, who believes that an abdominal anus will make life intolerable. The one-stage abdominoperineal resection for rectosigmoid and rectal lesions is still the operation of choice for the majority of surgeons. With no other procedure can 50 or more lymph nodes be removed with the lesion—nodes which may already be the seat of metastasis and which provide the pool for further spread subsequent to operation.

We must do everything possible to diagnose these lesions earlier; to increase operability; to make our resections more extensive; to think less of saving the rectum and more of curing the patient. More extensive resections are possible in better prepared patients. The incidence of infection subsequent to resection and anastomosis has been materially reduced by adjuvants to operative therapy. We must think less and less of the immediate operative mortality and more of 5-year and longer cures. Few fields of the surgery of malignant disease give such hope for the future as does this one. Ours is a great responsibility.

869434°—50——2

# Malignant Neoplasms of the Skin

ROBERT E. COKER, JR., *Lieutenant Commander (MC) U. S. N.*[1]

COMPARISONS of the incidence of diseases in the Navy and in the civilian population must always be made with caution. Among the reasons for this are (*a*) the differences in population structure—the Navy being made up largely of healthy young white men and (*b*) the differences in the completeness of reporting of disease incidence. In nonreportable diseases, estimates of incidence among civilians can usually be made only from death rates. Such estimates are often likely to go far afield.

In 1937 Peller and Stephenson (*1*), using death rates among civilians, concluded that the incidence of skin cancer in the Navy was eight times that of comparable civilian groups. On the assumption that Navy men were exposed to sunlight to an abnormal degree, a causal relationship between the two was postulated. This has been widely cited (*2*) (*3*) in support of the idea that exposure to sunlight plays a large part in the causation of skin cancer.

There has been a growing interest in malignant disease as a public health problem since the work of Peller and Stephenson, and figures on the incidence of neoplasms for selected civilian groups are now available. This makes it possible to make more direct comparisons between Navy personnel and civilian populations. Among the most reliable information available for large civilian groups is that based on the National Health Survey conducted from 1937 to 1939 and published by Dorn (*4*). These figures have been used to determine the difference, if any, between Navy and civilian experience with malignant neoplasms of the skin.

## PROCEDURE

The incidence rates for skin cancer per 100,000 population by 5-year age groups for men in the United States as a whole, given by Dorn, were applied by ages to the average strength of the Navy and Marine Corps for the years 1946 and 1947 combined. The sum of these products is the number of cases that would be expected to occur in the Navy if the rates for men in the country at large were operating in the Navy. An idea of the difference in experience between the two groups may be obtained by comparing this expected number with

[1] Medical Statistics Division, Bureau of Medicine and Surgery, Department of the Navy.

the actual number of cases occurring in the Navy during those years. This same procedure was also followed for malignant neoplasms of all sites. The ages considered were confined to the 5-year groups between 20 and 60 because this was the only area in which comparability of age-grouping could be obtained. It is believed that differences at the two ends of the age scale would be insufficient to make much of a change in the results. The rates for the younger ages are very low, and the Navy population over age 60 is very small.

## DISCUSSION

The reported and the expected incidence for malignant tumors of the skin in the entire Navy and Marine Corps for the years 1946 and 1947 combined are shown in table 1, and for malignant tumors of all sites in table 2. For skin tumors, the reported incidence of 157 cases is approximately twice the 77 cases which would have been expected if the rates for men in the United States in general had been effective in the Navy. This is in contrast to the eightfold difference estimated from the earlier data. In neoplasms of all sites the difference is almost as great; the reported incidence of 707 is 1.75 times the expected 400 cases.

TABLE 1.—*Malignant neoplasms of the skin, reported incidence and expected incidence—Navy and Marine Corps, 1946–47*

| Age | Average strength, Navy and Marine Corps | Incidence rate per 100,000 men, United States [1] | Expected cases, Navy and Marine Corps | Reported cases, Navy and Marine Corps |
|---|---|---|---|---|
| 20 to 24 years | 405, 595 | 3.4 | 27.6 | 40 |
| 25 to 29 years | 134, 057 | 5.2 | 13.9 | 26 |
| 30 to 34 years | 56, 825 | 8.9 | 10.1 | 27 |
| 35 to 39 years | 29, 516 | 11.0 | 6.5 | 19 |
| 40 to 44 years | 14, 151 | 22.6 | 6.4 | 14 |
| 45 to 49 years | 7, 998 | 34.4 | 5.5 | 15 |
| 50 to 34 years | 3, 400 | 63.1 | 4.3 | 8 |
| 55 to 59 years | 1, 302 | 96.2 | 2.5 | 8 |
| Total | 652, 844 | | 76.8 | 157 |

[1] See reference (4).

TABLE 2.—*Malignant neoplasms, all sites, reported incidence and expected incidence—Navy and Marine Corps, 1946–47*

| Age | Average strength, Navy and Marine Corps | Incidence rate per 100,000 men, United States [1] | Expected cases, Navy and Marine Corps | Reported cases, Navy and Marine Corps |
|---|---|---|---|---|
| 20 to 24 years | 405, 595 | 16.2 | 131.4 | 258 |
| 25 to 29 years | 134, 057 | 27.9 | 74.8 | 141 |
| 30 to 34 years | 56, 825 | 42.5 | 48.3 | 84 |
| 35 to 39 years | 29, 516 | 60.4 | 35.7 | 65 |
| 40 to 44 years | 14, 151 | 126.4 | 35.8 | 47 |
| 45 to 49 years | 7, 998 | 213.9 | 34.2 | 58 |
| 50 to 54 years | 3, 400 | 367.8 | 25.0 | 37 |
| 55 to 59 years | 1, 302 | 581.5 | 15.1 | 17 |
| Total | 652, 844 | | 400.3 | 707 |

[1] See reference (4).

The figures shown here cannot, for various reasons, be interpreted as showing the absolute relationship between the incidence of malignant tumors in the general population and in the Navy. The populations and periods of time involved are not completely comparable. Furthermore the Navy figures are the result of cases reported routinely while the civilian figures are based on a single survey. For these reasons, no attempt has been made to compute rates for the two groups for purposes of comparison. However, it is believed that the information examined is sufficient to support the conclusion that the incidence of skin cancer in the Navy is very little, if any, higher than in civilian groups of comparable age and sex.

There are numerous reasons which seem adequate to explain the apparent incidence of malignant growths to be greater for Navy personnel than for civilian groups, when actually this is not true. The men are subjected to regular physical examinations at frequent intervals. Complete medical care, including facilities for the microscopic examination of tissue, is readily available at no expense. Therefore, it is likely that the percentage of undiagnosed cases in the Navy is very low, while in general population it is known to be fairly high. Further, with rare exceptions, every diagnosed case in the Navy is reported. And, finally, if any bias is introduced by the difference in time of some 8 or 9 years between the Navy and civilian figures, that bias would work to make the Navy figure higher since there is evidence that the incidence of malignant tumors is increasing in the general population.

For these reasons it may be concluded that the apparent high incidence of malignant tumors in the Navy does not reflect the true state of affairs. In fact, there is no reason to believe that naval personnel develop tumors with greater frequency than similar persons in other occupations. The reported incidence of skin cancer in the Navy exceeds the expected incidence in about the same ratio as the Navy experience with malignant neoplasms of all sites exceeds that expected from civilian rates. This fact sustains the conclusions that more complete reporting and not environmental influences result in higher incidence of skin malignancy in the Navy.

### REFERENCES

1. PELLER, S., and STEPHENSON, C. S.: Skin irritation and cancer in the U. S. Navy. Am. J. M. Sc. **194**: 326–333, Sept. 1937.
2. BLUM, H. F.: Sunlight and cancer of the skin. J. Nat. Cancer Inst. **1**: 397–421, Dec. 1940.
3. VIEWAGER, V.: Sunlight is less important than you think. Hygiea **26**: 548, Aug. 1948.
4. DORN, H. F.: Illness from cancer in the United States. Pub. Health Rep. **59**: 33–48, Jan. 1944.

# Carcinoma of Lung

## Report of a Case Simulating Pleural Mesothelioma

LESTER W. FIX, *Lieutenant (MC) U. S. N.*[1]

SINCE the diagnosis of mesothelioma as a primary neoplastic lesion was first suggested (*1*) it has not been generally accepted and repeated reports have appeared (*2*) (*3*) (*4*) criticizing such a disease entity. The early literature presents too sketchy an account of both gross and histologic pathologic changes upon which to establish the diagnosis of mesothelioma. Geschickter (*5*) presented some interesting cases but Willis (*2*) failed to accept any of these as proved cases. Likewise many other authors (*2*) (*3*) (*4*) accepted only rare cases proved beyond question of doubt. Castleman (*6*) accepted one case and stated that this was the first case in which he believed there was actually such a tumor as a mesothelioma. Many other authors have presented single and multiple cases (*7*) (*8*) (*9*) (*10*) of supposed mesothelioma but critical analysis finds most of them lacking conclusive evidence on which to base the diagnosis. Indeed many of them prove to be a tumor other than mesothelioma. Certainly cases without autopsy protocol cannot be accepted as mesothelioma; these are either carcinomas of the lung or metastatic prostatic carcinomas. Because of this failure of general recognition, a case (seen at the U. S. Naval Hospital, Great Lakes, Ill.) is presented which demonstrated (*a*) the cellular structure sometimes described as typical of mesothelioma, (*b*) a clinical picture suggestive of mesothelioma, and (*c*) gross autopsy findings somewhat characteristic of mesothelioma, but conclusive microscopic evidence of the diagnosis of carcinoma of the lung.

### CASE REPORT

*History.*—A 19-year-old white man, not appearing acutely ill, fairly well-developed and well-nourished was admitted with the chief complaint of pain in the right chest posteriorly and slight chronic cough for almost 1 year. The past history was noncontributory except for a fractured fifth rib, 1 year previously; this fracture was not located near the site of the present complaint. Physical examination revealed normal temperature, respirations, and blood pressure. The pulse rate was 128. Dullness and decreased breath sounds were present over the

[1] U. S. Naval Hospital, USNTC, Great Lakes, Ill.

*Figure 1.*

*Figure 2.*

right posterior thoracic region. Roentgenograms taken in May 1948 (while in civilian status) showed a peripheral thoracic shadow (fig. 1). Roentgenogram (Navy) in January 1949, showed a mass in the right posterior superior thoracic cavity extending from the fourth to the seventh rib and measuring 11×6½ cm. (fig. 2). There was some erosion of the fourth and fifth ribs. Laboratory examinations showed: complete blood counts, urinalysis, Kahn test, Bence Jones protein, and albumin globulin ratio, all within normal limits. The sedimentation rate was elevated.

*Course.*—An exploratory thoracotomy was done and a sheet of tumor investing the right upper posterior lung was found. The pleura was considerably thickened in the area of the fourth and fifth ribs by tumor which had eroded these ribs. Surgical extirpation was impossible because of extension over the lung surface. A specimen for biopsy was taken and the surgical site was closed. The biopsy showed neoplastic tissue compatible with a mesothelioma and was identical with the lung tumor described later in the pathological report. The slides were reviewed by numerous experienced consultants who, for the most part, concurred with this impression. The patient's immediate postoperative course was stormy. High-voltage roentgen therapy was given and the patient improved enough to go on convalescent leave. Study of the roentgenograms when he returned showed a decrease in the size of the mass (fig. 3). The consensus was that this was a mesothelioma of the pleura although there was not

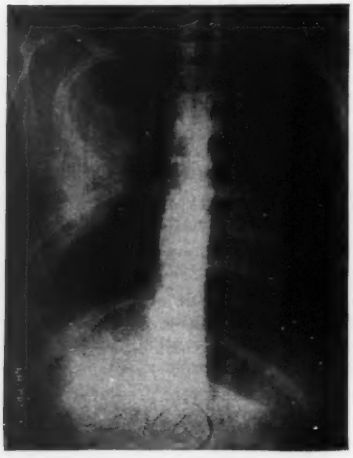


*Figure 3.*

complete accord in this diagnosis. The patient's course was progressively downhill and 12 February 1949, 5 months after his original admission to the hospital, he suddenly died.

*Pathological report*

*General description.*—The body is that of a thin, emaciated white man measuring 5 feet 2 inches and weighing approximately 90 pounds. There is a distinct discoloration of the right thorax.

*Cranial cavity.*—The brain weighs 1,640 gm. In the left frontal area there is a 5 cm. fairly well delineated, deep reddish-purple mass replacing most of the brain tissue. This mass is completely surrounded by a yellow, firm capsule of cerebral tissue. The mass shells out of the lumen with ease. In the left cerebellum there is a lesion 2 cm. in diameter, composed of soft white tissue. This tumor tissue is similar to that seen in the left lung. The fourth ventricle is noticeably dilated.

*Thoracic cavity.*—The bony cage presents a large mass which is firm and, in some areas, calcified, involving the third, fourth, fifth, and sixth ribs about 2 cm. from the vertebral origin. This tumor has resulted in some destruction of the fourth and fifth ribs. The mass extends into the pleural cavity and is firmly adherent to and infiltrating into the right lung. There is no free fluid in the left pleural cavity. The pleural cavity on the right is obliterated by tumorous adhesions. The pericardium is normal.

*Lungs.*—A thin neoplastic sheet covers almost the entire surface of the right lung. The lung is shrunken, firm, and almost completely nonaerated. In the outer margin, continuous with the mass previously described (see thoracic cavity), is a large soft white mass of neoplastic tissue. Cut sections of the lung show a homogeneous reddish-pink, firm fibrous pulmonary parenchyma. There is a sharp line of demarcation between the tumor and the pulmonary parenchyma. The left lung is mottled and grayish-purple. The cut sections reveal diffuse hemorrhage throughout the entire pulmonary parenchyma on this side. Undoubtedly there was rupture of some small vessel into a bronchiole resulting in massive intrapulmonary hemorrhage. The trachea is essentially normal. The right bronchus is essentially normal. On gross dissection there is no tumor within the major or minor bronchioles. The left bronchus, from the primary bronchus to the alveoli, is filled with recent fluid hemorrhage. The mediastinal lymph nodes are relatively normal in size but some of them are replaced by tumor. Dissection of the bronchi, bronchioles, and smaller ramifications of the bronchial tree fail to reveal any neoplasm.

*Microscopic description of major organs*

*Lungs.*—Sections through the lungs, underlying the neoplastic tissue described in the gross examination, show neoplastic tissue involving visceral pleura but sharply demarcated from the pulmonary parenchyma. Beneath this neoplastic tissue the pulmonary tissue shows considerable atelectasis with some dilation and congestion of the vessels. There are large numbers of pigment-filled macrophages scattered throughout the collapsed alveolar lumina. There is no evidence of invasion or infiltration of the neoplastic tissue beyond the visceral pleura of the lungs. The visceral pleura is extremely thickened and replaced in most areas by neoplastic tissue. This neoplastic tissue is composed of medium-sized polyhedral cells. These cells have large vesicular nuclei which are quite variable in shape. Many of the nuclei have multiple nucleoli which are likewise bizarre in shape. Mitotic figures are fairly frequent. The cyto-

plasm of these cells is fairly abundant and eosinophilic (fig. 4). Occasional papillary arrangement on a fine fibrous stroma is seen. The microscopic picture is identical with that seen in certain cases of primary hilar bronchogenic carcinoma. Study of many additional sections through the junction of the tumor mass in the underlying lung reveal a sharp line of demarcation between the pulmonary parenchyma and the visceral pleura with no tumor invading or infiltrating the pulmonary parenchyma. Multiple sections of the lungs reveal diffuse, hemorrhagic infiltration throughout the alveolar lumina. The blood vessels are greatly dilated and congested. There is some fibrosis of the interstitial tissue in the right middle lobe. The bronchi and bronchioles are filled with red blood cells. Repeated sections of lung finally resulted in the demonstration of histologic area of neoplastic tissue identical with the main pleural mass and with the metastatic lesions. This lesion was a 2-mm.-in-diameter area of neoplastic tissue almost completely filling the lumina of a tiny bronchiole. The neoplastic tissue is continuous with essentially normal bronchial epithelium in one area (fig. 5). Except for this microscopic lesion there is no other evidence of tumor in the lung.

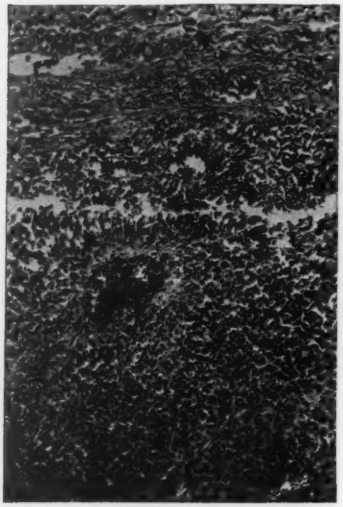


*Figure 4.*

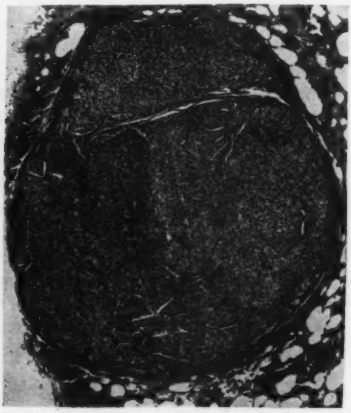


*Figure 5.*

*Adrenal.*—In the adrenal there is a well-delineated mass of neoplastic tissue identical with that found in the lung.

*Bone marrow.*—Sections show bony spicules which are fairly well preserved. In some areas, scattered throughout the bone marrow, there are small islets of neoplastic tissue composed of cells the same as those found in the lung.

*Lymph nodes.*—Sections through the mediastinal lymph nodes reveal neoplastic tissue replacing the lymph nodes. The cells in this neoplastic tissue are identical with those previously described.

*Brain.*—Sections of the neoplastic lesion described in the brain reveal tumor identical with that in the lung. The adjacent cranial tissue is compressed but not invaded by the neoplastic tissue. The brain elsewhere shows some neuronedema.

*Diagnoses.*—The final anatomical diagnoses were: (*a*) subpleural primary carcinoma of right lung; (*b*) extensive pleural metastases with carcinomatous obliteration of the pleural cavity (simulating mesothelioma); (*c*) carcinoma metastases to mediastinal lymph nodes, adrenal, bone marrow, and brain; and (*d*) pulmonary hemorrhage in the left lung.

## DISCUSSION

If this case was mesothelioma it presents the problem of whether mesothelioma can metastasize to the lungs. The demonstration of a lesion in one bronchiole which is identical with the neoplastic tissue elsewhere and the demonstration of neoplasm in the mediastinal lymph nodes, pleura, bone, and brain leads to the conclusion that this was a primary neoplasm of the lung. Despite the small size of the original lesion and the tremendous size of the pleural metastases, the author believes that this was a primary bronchogenic carcinoma. This is based on the high incidence of bronchogenic carcinoma, in contrast with the low incidence of mesothelioma, the sites of metastases typical for bronchogenic carcinoma, and the microscopic contiguity of the tumor in the bronchiole with normal epithelium.

## SUMMARY

An interesting case of carcinoma of the lung is presented in which both the operative and gross autopsy diagnoses were mesothelioma of the pleura. Multiple microscopic sections from about 40 different areas finally demonstrated a small lesion in a minor bronchiole which was identical with the other neoplastic tissue; this was regarded as the primary lesion. Prior to this evidence the author was convinced that this was indeed a case of mesothelioma. Serial sectioning of many blocks may be necessary before the diagnosis can be definitely established in unusual cases which present a histologic picture simulating the description of mesothelioma of the pleura. In our case such a procedure resulted in the diagnosis of carcinoma of the lung arising from a tiny bronchiole rather than a mesothelioma as previously suspected from both clinical and gross autopsy appearance.

### REFERENCES

1. WAGNER, E.: Das tuberkelahnliche lymphadenoma. Arch. f. Heilk. 11: 497, 1870.
2. WILLIS, R. A.: Pathology of Tumors. The C. V. Mosby Co., St. Louis, Mo., 1948.
3. MOORE, R. A.: Textbook of Pathology. W. B. Saunders Co., Philadelphia, Pa., 1945.
4. FOOTE, N. C.: Identification of Tumors. J. B. Lippincott Co., Philadelphia, Pa., 1948.
5. GESCHICKTER, C. F.: Mesothelial tumors. Am. J. Cancer 26: 378–396, Feb. 1936.

6. Massachusetts General Hospital, Case 33111: Mesothelioma of pleura and pericardium, with metastases to right lung and retroperitoneal lymph nodes. New England J. Med., 236: 407–412, March 13, 1947.
7. ZINNINGER, M. M.: Tumors of wall of thorax. Ann. Surg. 92: 1043–1058, Dec. 1930.
8. KLEMPERER, P., and RABIN, C. B.: Primary neoplasms of pleura; report of five cases. Arch. Path. 11: 385–412, March 1931.
9. ZECKWER, I. T.: Mesothelioma of pleura. Arch Int. Med. 34: 191–205, 1924.
10. PIATT, A. D.: Primary mesothelioma (endothelioma) of pleura; case report. Am. J. Roentgenol. 55: 173–180, 1946.

BIBLIOGRAPHY

1. KARSNER, H. T.: Human Pathology. 6th edition. J. B. Lippincott Co., Philadelphia, Pa., 1943.
2. EWING, J.: Neoplastic Diseases. 4th edition. W. B. Saunders Co., Philadelphia, Pa., 1940.
3. ACKERMAN, L. V., and DEL REGATO, J. A.: Cancer: Diagnosis, Treatment, and Prognosis. The C. V. Mosby Co., St. Louis, Mo., 1947.

## Gentleman and Scholar [1]

On a recent visit to Goettingen, the seat of the venerable Georg-August University, I met the renowned pathologist, Gg. B. Gruber, who formerly held the chair of pathology at the university, and is now retired. He told me the following story: In April 1945, when the American troops entered Goettingen, an American medical officer entered the Pathological Institute looking for a place to billet his men. He examined every room in the Institute, and was attracted by the gallery of pictures of pathologists. When he noticed among these a picture of Rudolph Virchow, he said: "A house in which the picture of Rudolph Virchow hangs, whom we Americans admire not only as a scientist but also as a democrat, shall not be occupied by the military." He gave a brisk military salute, and left. The Pathological Institute of Goettingen remained unoccupied.

—*Robert Wartenberg, M. D.*

[1] California Med. 72: 33, Jan. 1950.

# Thyroid Cancer in Young Adults

## Report of Four Cases

ROBERT B. STROTHER, *Commander (MC) U. S. N.*[1]
LESTER W. FIX, *Lieutenant (MC) U. S. N.*[1]

A GREAT variance in the incidence of carcinoma of the thyroid gland exists in the literature. Some of this occurs as a result of the different criteria used for the diagnoses of malignancy of the thyroid gland. In addition deviations occur in the incidence in different age, sex, and race groups as well as in the number and duration of the lesions.

Several articles have stressed the high incidence of cancer in nodular goiter (*1*) (*2*) (*3*) (*4*) and some authorities have stated that they believe all carcinomas of the thyroid to be derived from fetal adenomas or aberrant thyroid tissue (*5*). However, the incidence varies considerably with different authors and with different types of lesions. Thus, Ward (*4*) found an incidence of 4.8 percent of thyroid cancer in nodular goiter while Cole et al. (*1*) found an incidence of 7.2 percent. Further breakdown of Cole's figures shows an incidence of 17.1 percent in nontoxic nodular thyroid masses and 24.0 percent in solitary nontoxic nodules. Warren (*6*) concurs in the higher incidence of carcinoma in solitary nodules and states that isolated nodules are 10 times more likely to be malignant than any other type of thyroid enlargement.

Opinion is divided as to geographic distribution. Ward (*4*) states that the incidence of malignancy is influenced by the degree of endemicity and the frequency of nodular goiter. On the other hand in a nongoitrous area such as Boston, Schlesinger et al. (*7*) report a much higher incidence (4.5 percent) of cancer in the thyroid nodules found at autopsy than Jaffé (*8*) found in Chicago (0.2 percent). It is interesting to note, however, that the high incidence reported by Cole et al. (*1*) was found in the same region in which Jaffé's studies were made. There is some conflict in the age incidence of carcinoma of the thyroid and many authors put the average incidence at about 40 years of age or more (*1*) (*11*). All agree that cases do occur in youth and this article is written to stress this point. There is general agreement

[1] U. S. Naval Hospital, USNTC, Great Lakes, Ill.

as to the incidence in the sexes and most authors concur that the ratio of carcinoma of the thyroid gland between men and women is about 1 to 4.

There is a striking difference in the figures for diagnosed cancer (*1*) (*2*) (*3*) (*4*) when compared with the very low incidence of cancer found at autopsy (*9*). One explanation is that given by Means (*10*), who states that patients having cancer are frequently sent home where their terminal course is spent and thus these do not appear in autopsy statistics. Means reports that in 1 year, eight of his patients died of cancer of thyroid, and only one of them died in the hospital.

In view of the wide variation in ideas concerning cancer of the thyroid gland it was considered desirable to report four interesting cases seen by the authors in a 9-month period at the U. S. Naval Hospital, Great Lakes, Ill. During this period there were only six thyroidectomies performed for nodular thyroid lesions.

## CASE REPORTS

*Case 1.*—R. J. N., a 19-year-old white man, was admitted complaining of a lump in the left thyroid region of about 5 years' duration. There had been no change in the size of the lesion, no weight loss, or change of appetite. He stated that he had always been slightly nervous.

Past history was irrelevant. He had lived in Maryland all his life.

*Physical findings.*—The patient was a well-developed, well-nourished young man with a firm nontender nodular enlargement of the left lobe of the thyroid gland. The nodule was irregular and appeared to involve the entire left lobe which was freely movable. There were three or four palpable nodes along the anterior border of the left sternocleidomastoid muscle which were of similar consistency, nontender, and freely movable.

*Laboratory examination.*—Blood counts and urinalyses were normal. Blood Kahn test was negative. Blood cholesterol was 228 mg. per 100 cc. blood. Roentgenograms of the chest, skull, pelvis, and long bones were negative.

An operation was performed on 13 May 1948. The entire left lobe of the thyroid gland was removed along with what appeared to be five small masses of aberrant thyroid tissue located along the anterior border of the left sternocleidomastoid muscle.

The pathologic examination revealed a mass, 6×4×4 cm., with a firm capsule. In one area there was a semisoft, greenish-white nodule 1 cm. in diameter. Cut sections showed greenish gelatinous material accompanied by three masses varying from 2.2 to 2.5 cm. in diameter which also showed greenish, gelatinous material on section. Microscopic study showed papillary cyst adenocarcinoma of the thyroid gland.

Tracer doses of radioactive iodine ($I^{131}$) were given. An additional lymph node containing thyroid tissue, along the sternocleidomastoid border, was discovered; this was removed and therapeutic doses of radioactive iodine were administered to produce a moderate myxedema.

The patient was in good health 1 year following surgery.

*Case 2.*—G. E., an 18-year-old white man, was admitted to the hospital for plastic repair of a disfiguring scar of the left cheek.

Past history was irrelevant. He had lived in North Dakota all his life.

*Physical examination.*—In addition to the scar on the face, the patient had a firm, nontender, discrete nodule about 1 cm. in diameter in the left lobe of the thyroid near the midline. The mass was freely moveable and not attached to adjacent tissues or skin. There were no other palpable nodules in the neck. The right testes had been removed previously for cryptorchism.

*Laboratory examination.*—Blood counts and urinalyses were normal. Blood Kahn test was negative. Blood cholesterol was 194 mg. per 100 cc. blood, and the basal metabolism rate was plus 4. Roentgenograms of the neck and chest showed no abnormalities.

At operation on 4 January 1949, a firm nodule was found almost completely replacing the left lobe of the thyroid; about nine-tenths of the left thyroid lobe was removed.

The postoperative course was uneventful.

Pathological examination showed a 2.7 cm., firm, fairly well encapsulated nodule. Cut sections showed a yellow-brown granular surface in the center of which there was a pearly-white area. Microscopic studies showed a papillary cyst adenocarcinoma secondary to a colloid adenoma (fig. 1).

*Case 3.*—J. O. G., an 18-year-old white man, was admitted to the hospital because of a mass in the neck.

*Past history.*—He stated that he had had this nodule for about 4 years. His family physician had prescribed iodized salt as a therapeutic measure 4 years previously. The patient continued this therapy periodically until his entrance into the Navy; he had noted a slight regression in the size of the nodule.

The patient had also noticed some sweating of the hands, palpitation, and, recently, slight dysphagia.

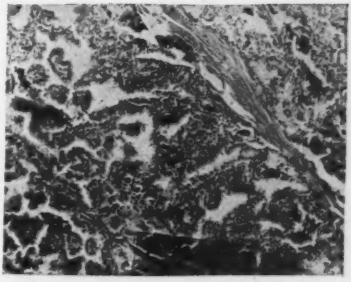


*Figure 1.—Case 2. Photomicrograph showing papillary cyst adenocarcinoma pattern (× 110).*

*Physical examination.*—The patient was a well-developed, well-nourished man. Only slight moistness of the palms was noted. A nodule was found in the thyroid region which measured about 3 cm. in diameter and was firm, nontender, not attached to skin or underlying tissue, and involved most of the isthmus of the thyroid gland.

*Laboratory examinations.*—Blood counts and urinalyses were normal. Blood Kahn test was negative. Blood cholesterol was 165 mg. per 100 cc. blood; the basal metabolism rate was minus 15. Roentgenogram of the chest and neck showed no evidence of abnormality.

An operation was performed on 15 February 1949, and a large, firm nodule was removed. The nodule involved only the isthmus and a major portion of the left lobe of the thyroid. The isthmus and left lobe of the thyroid gland were removed.

The patient's course was uneventful.

Pathologic examination revealed a 5½ cm. irregular nodule. The capsule was tense, but appeared grossly intact. Cut sections revealed homogeneous, dense, pinkish-white tissue. Microscopic examination revealed a papillary adenocarcinoma.

*Case 4.*—I. B., a 25-year-old white woman first noted an enlarging mass in the region of the left thyroid in November 1948. This had developed into a "thumb-sized mass almost overnight." This mass continued to grow and in December 1948 there was a 2.0 cm. lesion in the left thyroid area.

*Past history.*—She had delivered a full term normal infant in June 1948. During her pregnancy she had gained considerable weight and she was given thyroid extract in an attempt to reduce her weight.

There was no evidence of previous thyroid disease and no familial history of thyroid disease.

*Physical examination.*—The patient was a well-developed, slightly obese young woman. A nodule was found in the left lobe of the thyroid gland. This measured 2 cm. in diameter, was soft, cystic, nontender, and was attached to the left lobe of the thyroid gland. There were no other abnormal masses in the neck.

*Laboratory examination.*—Blood counts and urinalyses were normal. Blood Kahn test was negative. Blood cholesterol was 210 mg. per 100 cc. blood. The basal metabolism rate was minus 6. Roentgenogram of the chest and neck showed no abnormalities.

*Figure 2.—Case 4. Photomicrograph showing the variations in cellular characteristics* (×100).

An operation was performed on 12 January 1949. A large cystic nodule and about seven-eighths of the left thyroid lobe was removed.

Pathologic examination showed a 5×3×2 cm. mass of thyroid tissue in which there was a well-encapsulated grayish-red nodule 2 cm. in diameter. Cut sections showed mottled red and white tissue arranged in whorls. In two areas the capsule was grossly penetrated by the reddish-white tissue. Microscopic examination revealed an admixture of types of carcinoma with a predominance of adenocarcinoma (fig. 2).

Before the diagnosis of cancer was made, the following criteria were met in all cases: (*a*) Definite histologic change characteristic of cancer; (*b*) evidence of capsular invasion; and (*c*) evidence of lymph and blood vessel invasion.

The Navy Tumor Registry reviewed the cases and concurred in the diagnoses.

## SUMMARY

Four cases of carcinoma of the thyroid gland are presented. The cases were in young persons, the oldest being 25 years of age. All presented single nodules of the thyroid gland. In two cases there was a known history of the presence of a thyroid nodule for 4 and 5 years respectively. In a third case the duration of the nodule was unknown, while in a fourth case the nodule was known to have been of very recent origin. All presented minimal or no symptoms. There was adjacent lymph node involvement in one case, while the others showed no local spread. These four cases of carcinoma of the thyroid out of six patients with nodules in the thyroid point out that carcinoma of the thyroid gland should be carefully considered in the diagnosis of thyroid masses in young men and women in the Navy.

A review of the literature reveals that malignant tumors of the thyroid are not rare and that they do occur in the younger age groups. It is of the utmost importance to emphasize the high incidence of malignancy in solitary nontoxic adenoma of the thyroid. This recognition, plus the decreased operative risk of thyroid surgery, makes the removal of single nontoxic adenomas mandatory. The youth, the occurrence in men, and the factor of a single nontoxic nodule are emphasized. The necessity for surgical removal of all single nontoxic nodules of the thyroid is stressed.

## References


1. Cole, W. H.; Slaughter, D. P.; and Rossiter, L. J.: Potential dangers of nontoxic nodular goiter. J. A. M. A. **127**: 883–888, April 7, 1945.
2. Cattell, R. B.: More optimistic approach to cancer of thyroid. West. J. Surg. **54**: 444–449, Nov. 1946.
3. Crile, G., Jr.: Cancer of thyroid. Postgrad. Med. **1**: 51–57, Jan. 1947.
4. Ward, R.: Malignant goiter. Surgery **16**: 783–803, Nov. 1944.
5. Gargill, S. L., and Lesses, M. F.: Diseases of thyroid gland. New England J. Med. **235**: 717–728, Nov. 14, 1946.
6. Warren, S.: Classification of tumors of thyroid, Am. J. Roentgenol. **46**: 447–450, Oct. 1941.
7. Schlesinger, M. J.; Gargill, S. L.; and Saxe, I. H.: Studies in nodular goiter; incidence of thyroid nodules in routine necropsies in nongoitrous region. J. A. M. A. **110**: 1638–1641, May 14, 1938.
8. Jaffé, R. H.: Variation in weight of thyroid gland and frequency of its abnormal enlargement in region of Chicago. Arch. Path. **10**: 887–894, Dec. 1930.
9. Vanderlaan, W. P.: Occurrence of carcinoma of thyroid gland in autopsy material. New England J. Med. **237**: 221–222, Aug. 14, 1947.
10. Means, J. H.: Thyroid and Its Diseases. 2d edition. J. B. Lippincott Co., Philadelphia, Pa., 1948.
11. Pemberton, J., and Lovelace, W. R., Jr.: Malignant lesions of thyroid. S. Clin. North America **21**: 1037, Aug. 1941.

# Administration of Procaine Intravenously

## V. In Medical Conditions[1]

HOWARD K. PEDIGO, *Major, MC, U. S. A.*[2]

IN GENERAL, procaine is used in medicine for the relief of pain. It apparently acts directly on the cells involved in diseased processes as well as on nerve endings and in addition exhibits a general antisympathetic action. In diseases of the skin, procaine has been used to treat contact dermatitis, exfoliative dermatitis, and in the pruritus of jaundice to control itching and irritation (*1*). In the first two instances procaine assists in the cure of the underlying condition by checking the spread of the irritation that results from scratching. It has been tried in the pruritus of infectious granuloma without success. Procaine has been so successful in relieving the urticarias associated with transfusion reactions that some advocate giving it in the transfused blood, if a patient gives a history of previous reactions. Procaine has been successfully used intravenously and intra-arterially to treat cellulitis in an extremity, based on the assumption that the resultant increase in the blood supply to the affected part would improve the nutrition of that part (*2*). Procaine infusion has been used to relieve the pain associated with herpes zoster (*3*). It has been used for pain in leprous ulcers of the legs (*4*), and has speeded the granulation of atrophic ulcers of the legs. When used to treat very recent frostbite and trench foot, the results were gratifying and the time spent in the hospital was reduced appreciably (*5*). However, this treatment was not of benefit when applied more than 24 hours after the injury was incurred.

The cardiologists have reported long periods of relief from the pain of angina pectoris in patients in whom no relief was obtained from the use of opiates and nitroglycerin (*6*). After a series of infusions of a 1-percent solution of procaine, most of these patients were symp-

[1] This concludes this series. Parts I and II were published in the December 1949 issue of the Bulletin of the U. S. Army Medical Department. Part III was published in the January 1950 issue of the U. S. Armed Forces Medical Journal, and Part IV was published in the February 1950 issue of the U. S. Armed Forces Medical Journal.

[2] Brooke General Hospital, Fort Sam Houston, Tex.

tom-free up to 6 months. A few patients with cardiac embolism of both sides of the heart have been treated by this means with apparent success (*7*). The results in the treatment of paroxysmal tachycardia are not conclusive.

Although many reports indicate failure in the use of procaine in asthma, most of the authors observed beneficial results after other medications had failed (*8*). The action seems to be a reduction of the sensibility of the vegetative nerve endings and relief of the bronchospasm. Procaine infusion has been remarkably successful in a large number of cases of dyspnea. The dyspnea of old age is apparently relieved because of the increase in cardiorenal efficiency and the reduction of the moderate pulmonary congestion that is usually present in such conditions (*9*). There are reports of the relief of the dyspnea associated with terminal tuberculosis and pulmonary carcinomatosis. It has been used in chronic and acute bronchitis with a reduction of the mucus expectoration as well as of the subjective symptoms. Other authors report an immediate functional improvement when it is used in pulmonary edema (*7*).

In acute thrombophlebitis, not only has relief of pain been reported, but also by means of venographic studies it has been demonstrated that procaine infusion has resulted in venous patency where none existed before. As an aid to diagnosis, it has allowed the exact area of phlebitis to be outlined by the radiopaque medium (*10*). In arteritis, increase in the warming of an extremity and in the pulsations of the affected artery has been frequently demonstrated and, at the Brooke General Hospital, intravenous procaine has been used for the relief of the headache associated with temporal arteritis. We have also used it in conjunction with lumbar sympathetic blocks to prolong the action of the block.

Procaine will not prevent anaphylaxis but will suspend some allergies (*11*). In full-blown serum sickness it has been used with success to relieve promptly the arthralgia and muscular spasm and as a palliative measure for the urticaria that accompanies this condition (*12*) (*13*) (*14*). It has been used with massive doses of neoarsphenamine (as much as 1.05 gm. in one injection) and other arsenical drugs to prevent reactions (*15*). However, some of the delayed toxic manifestations did occur in a few of these patients, but not the icterus, polyneuritis, and dermatitis of the sensitivity reaction.

A major use in both medicine and surgery is in relieving reflex anuria (*16*) (*17*) (*18*). The use of procaine in this condition is well recognized and fairly widespread. There are instances when a complete anuria has been relieved in a few hours. It is used with success in the anuria and oliguria associated with sulfonamide and arsenical therapy (*19*). There has been some success in the treatment of acute

but not chronic nephritis with procaine infusion. It acts as an efficient diuretic in hypertensive cardiovascular renal disease. It does not, however, affect the blood pressure. The action in all these conditions is an improvement of the glomerular circulation with a relief of the vasoconstriction when present. It is beneficial in the treatment of orchitis and epididymitis (*7*). It has been used extensively for the relief of arthritic pain, pain of articular origin, sciatica, lumbosacral pain, and most other pains of diseased bone and periosteum (*20*). In rheumatoid arthritis, it is as specific as the salicylates are in rheumatic fever. In osteo-arthritis, definite improvement has been demonstrated by roentgenogram with complete subjective relief. The pain and muscular spasm of other types of arthritis may be temporarily relieved. Little or no relief is given in most cases of primary or metastatic carcinoma of the bone. Procaine infusion gives relief to patients with a continuous tinnitus aurium (*21*) (*22*). Those with a pulsating type of tinnitus were not relieved. There was no improvement in the hearing of any of the subjects. The time of maximum effect was at most only a few hours. It has been assumed that the drug action here is on the central nervous system.

Although procaine infusion has been tried on more varied types of disease than any other drug of recent history, it is not a universal pain reliever. Its complete action and limitations have not yet been entirely worked out, and since in some patients the therapeutic dose and the toxic dose are so close, it should only be used under the direct supervision of a person well-acquainted with the toxic manifestations and well-equipped to handle the occasional convulsive reaction or other form of toxicity that may occur. It has been found that when a convulsion or syncope occurs during the administration of a procaine infusion, the immediate injection of 3 to 5 cc. of 2.5 percent pentothal sodium solution and oxygenation will promptly relieve all the symptoms.

### References

1. Deuil, R., and Audoly, P.: La novocaïne intraveineuse contre le prurit des icteriques. Paris méd. **33**: 314–315, Nov. 30, 1943.
2. Leriche, R.: A propos de la thérapeutique par voie artérielle dans les infections et en dehors de l'infection. Mém. Acad. de chir. **64**: 220–224, Feb. 9, 1938.
3. Ravina, A.: Traitement du zona et des algies post-zonateuses par la novocaïne intraveineuse. Presse méd. **53**: 124, March 10, 1945.
4. Guadagnini, M.: Tratamiento de las ulceras leprosas por la novocaina endoarterial. Rev. argent. dermatosif. **29**: 278–282, Dec. 1945.
5. Berthelemy, A.: Une application thérapeutique nouvelle, la novocaïne intraveineuse, aspect chirurgical de cette médication. Progrès méd. **73**: 413–415, Dec. 10, 1945.
6. Ameuille, P.: La novocaïne intraveineuse. Progrès méd. **73**: 407–413, Dec. 10, 1945.

7. HAZARD, R.: Notions actuelles sur les actions physiologiques de la novocaïne et sur ses applications thérapeutiques. Progrès méd. **73:** 403–407, Dec. 10, 1945.
8. DURIEU, H.; DE CLERCQ, F.; and DUPREZ, A.: Traitement de l'asma par la novocaïne intraveineuse. Acta clin. belge. **1:** 150–159, March-April, 1946.
9. BRETON, A., and GUIDOUX, A.: La dyspnée de ieillard; son traitement par les injections intraveineuses de novocaïne. Paris méd. **30:** 302–303, Sept. 30, 1942.
10. DECOURT, J.: Les injections locales de novocaïne dans le traitement de certaines algies de caractère sympathique. Paris méd. **109:** 423–429, Dec. 3, 1938.
11. STATE, D., and WANGENSTEEN, O. H.: Procaine intravenously in treatment of delayed serum sickness. J. A. M. A. **130:** 990–995, April 13, 1946.
12. APPELBAUM, E., ABRAHAM, A.; and SINTON, W.: Case of serum sickness treated with procaine intravenously. J. A. M. A. **131:** 1274–1275, Aug. 17, 1946.
13. DRESSLER, S., and DWORK, R. E.: Reactions to penicillin; procaine hydrochloride intravenously in treatment of reactions similar to serum sickness. J. A. M. A. **133:** 849, March 22, 1947.
14. BRETON, A., and GUIDOUX, A.: Le mecanisme nerveux de l'anaphylaxie et de l'allergie. Sa dissociation par la novocaïnisation intraveineuse. Intérêt pratique de la question. Progrès méd. **70:** 412–414, Oct. 10, 1942.
15. VANHAECKE, E.; BRETON, A.; and GUIDOUX, A.: Arsénothérapie massive avec novocaïnisation intraveineuse préalable. Soc. méd. du Nord. June 26, 1942, reported in Presse méd. **50:** 601, Sept. 26, 1942.
16. AUGUSTE, C., and HERAUD, M.: La novocaïne intrapéritoneale dans le traitement des manifestations douloureuses de l'ulcère gastro-duodénal. Soc. méd. du Nord. Nov. 26, 1943; reported in Presse méd, **52:** 91, March 18, 1944.
17. HILLEMAND, P.; MONTALANT, P.; BARRÉ, Y.; and LEGOARANT DE TROMELIN, G.: Deux cas d'anurie traités et guéris par des injections intraveineuses de novocaïne. Bull. et mém. Soc. méd. d. hôp. de Paris **60:** 182–184, May 5, 1944.
18. LANGERON, L.: Note sur l'utilisation de la scurocaïne intraveineuse dans les néphropathies, son action diuretique probablement par mècanisme vasomoteur. Bull. et mém. Soc. med. d. hôp. de Paris **60:** 13–14, Jan. 14, 1944.
19. QUEMARES. J.: Anuries aiguës: Intraveineuse novocaïne. Thesi méd. Paris **290:** 1946.
20. LANGERON, L.; PAGET, M., and MICHAUX, P.: Anurie par le sulfathiazol, Anurie sécrétoire. Blocage tissulaire. Action remarquable de la scurocaïne intraveineuse. Bull. et mém. Soc. méd. d. hôp. de Paris **62:** 58–61, Jan. 18, 1946.
21. GRAUBARD, D. J.; KOVACS, J.; and RITTER, H. H.: Management of destructive arthritis of the hip by means of intravenous procaine. Ann. Int. Med. **28:** 1106–1116, June 1948.
22. LEWY, R. B.: Treatment of tinnitus aurium by the intravenous use of local anesthetic agents. Arch. Otolaryng. **25:** 178–183, Feb. 1937.
23. BÁRÁNY, R.: Die Beeinflussung des Chrensausens durch intravenös injizierte Lakalanästhetica. Acta oto-laryng. **23:** 201–203, 1935.

# Combined Coarctation and Rupture of the Aorta

ALFRED BRODY, *Captain, MC, U. S. A.*[1]
HERWIG HAMPERL, *M. D.*[2]

A REVIEW of the literature revealed 38 cases of combined coarctation and rupture of the aorta (*1*) (*2*) (*3*) (*4*) (*5*) (*6*) (*7*) (*8*) (*9*). A detailed microscopic description was given in only 15 case reports and in no case was the rupture healed. Coarctation of the aorta is a narrowing or stenosis usually occurring near or proximal to the junction with the ductus arteriosus. Bonnet (*10*) divided these cases into infantile and adult types. In the infantile type the constriction is between the subclavian artery and ductus arteriosus and a patent ductus distributes venous blood through the descending aorta. This is probably caused by a failure of fusion of the two parts of the aorta in early fetal life and is usually incompatible with more than a few weeks of extrauterine existence. In the adult type, the constriction is at about the level of the ductus arteriosus which is completely obliterated. Arterial blood reaches the descending aorta through a greatly enlarged collateral circulation, detection of which is one of the two clinical findings possible in these cases. The proximal arteries are the internal mammary, the descending branches over and under the scapula, and the anterior spinal. The distal arteries are the intercostals, and the inferior epigastrics. This type may be caused by a continuation of the obliterative endarteritis of the ductus into the adjacent aorta (*9*).

Death occurs from 3 to 72 years of age (average 32) usually as a result of the hypertension of the proximal segment and its branches (*7*). In 30 percent of the cases, death results from cardiac failure; in 20 percent from rupture of the aorta; in 10 percent from cerebral hemorrhage; and in about 8 percent from endocarditis. In the remainder, death is unrelated to the coarctation. Bicuspid aortic valves occur in almost 18 percent of the cases (*9*).

[1] Valley Forge General Hospital, Phoenixville, Pa.
[2] Algemeines Krankenhaus, Salzburg, Austria.

The difference in blood pressure between the proximal and distal segments measured in the arm and leg respectively furnishes the second chief clinical criterion of this condition. One of the chief reasons for recognizing this type of hypertension is the possibility of cure by resection of the stenosed portion of the aorta (*11*) (*12*) (*13*).

Idiopathic cystic medial necrosis of the aorta (*11*) (*14*) is a condition of unknown cause increasing in incidence and severity with age and predisposing to dissecting aneurysm and rupture of the aorta. While such a rupture may occur without these changes in patients with severe crushing injuries, most of such cases and all so-called spontaneous ruptures are preceded by weakening of the aortic wall by the deposition of a homogeneous, pale-staining, basophilic, acellular material in the media of the aorta between the muscle cells and about the elastic fibers. This microscopic finding may be present without gross changes or it may be present with aortic rupture, typically a transverse tear 1 to 4 cm. in length and about 1 cm. above the valves. This may extend into the pericardium permitting cardiac tamponade by blood, or the rupture, after extending about two-thirds through the media, may dissect along the length of the aorta to rupture at some other point, either into or out of the aorta. Healing of this type by thrombus formation or endothelialization is rare (*9*).

The entire condition is 3 to 9 times as frequent in men as in women and is found most frequently between the ages of 45 and 60. Usually rupture or dissecting aneurysm occurs in association with hypertension. Clinically, a shocklike state, resembling an acute coronary occlusion, frequently with maximum pain in the back is followed by relief, death from cardiac tamponade, or evidence of hemorrhage into the tissues along the aorta. Occasionally, symptoms are partly or mainly caused by narrowing or closure of the ostii of the aortic branches by pressure from within the aortic wall. Vessels other than the aorta are rarely involved in rupture or dissecting aneurysm.

## CASE REPORT

A 55-year-old male laborer, first admitted to a hospital in September 1947, complained of dyspnea, cyanosis, and weakness which had begun following an attack of influenza early in the year and had increased. His blood pressure was 200/85 in both arms. His pulse was soft with a rate of about 80. Physical examination showed vesicular and bronchial piping râles throughout the chest, most distinct in the bases. The heart was greatly enlarged to the left. Aortic and tricuspid systolic and diastolic as well as soft mitral diastolic murmurs were heard. The liver was enlarged three fingerbreadths below the costal margin, but there was no edema of the extremities. A roentgenogram of the chest showed a generalized enlargement of the heart. Electrocardiography was not possible and the urine was normal. The patient was given strophanthin, made a rapid recovery from most of his symptoms and was discharged on a maintenance dosage of digitalis.

Late in November 1947, he was readmitted with a severe recurrence of his previous symptoms plus severe generalized edema of legs, thighs, back, arms, and abdomen. The urine at this time contained 4 plus albumin. During a 5-month course of treatment the patient was kept in cardiac compensation only by the vigorous use of mercurial diuretics, as well as digitalis. On 6 April 1948 he was transferred to another hospital in a state of cardiac decompensation, with a diagnosis of hypertensive cardiovascular disease. He died of pulmonary edema less than 24 hours after admission, despite all therapy.

At autopsy the heart was found to be almost twice the normal size. The entrances to both chambers were dilated. The wall of the left chamber was 17 mm. thick in the midsection. The endocardium was white. The trabeculae were flattened and changed to white cords especially in the region of the posterior wall. In the heart muscle, at this site, there was an extensive white scar. The medial mitral valve was thickened by fibrous tissue. The chordae tendineae were thin. The wall of the right chamber was 6 mm. thick. The chordae tendineae of the tricuspid valve were thin. Between the strongly developed trabeculae in the region of the apex rounded parietal thrombi were found. The pulmonary valves were thin. The circumference of the pulmonary artery just above the valves was 8 cm. The right auricle was dilated. In the auricle, numerous parietal half-rounded projecting thrombi were present. The foramen ovale was closed. In the intima of the coronary arteries there were many fatty and fibrotic plaques which narrowed the lumen.

At the origin of the aorta there were only two half-moon-shaped valves, one behind and one in front. At the right commisure the two valves formed a spur 15 mm. long that was directed upward. In the region of the forward-facing valve, the right coronary artery arose about 1 cm. to the left of the spur and the left coronary artery arose 1 cm. to the right of the other commisure. The forward valve showed comblike chalky projections directed outward. Both sinuses of Valsalva were dilated almost to hemispheres. The valves, themselves, were tightly stretched. The circumference of the aorta at the level of the valves was 13 cm. The inner wall of the ascending aorta showed many flat, yellow plaques. The lumen was dilated backward and to the right so that the circumference, in this part, was enlarged to about 14 or 15 cm., then, shortly before the origin of the vessels of the neck, returned to 7 cm. Above the posterior valve, the aortic wall showed an almost T-shaped tear of the inner layers of the wall. Between the edges of the tear a smooth internal covering over white connective tissue was seen. The crosspiece of the T was about 1 cm. above the base line of the valves and the stem of the T ran zig zaggedly upward. The crosspiece was 4 cm. and the upward running stem about 6 cm. long.

The circumference of the inominate artery was 5 cm., of the right and left subclavian arteries 4.6 cm., and the diameter of both carotids was about 2.5 cm. The internal mammary arteries measured 2 cm. at the level of the third rib. There were fatty plaques in the intima of all these vessels. About 2.5 cm. below the origin of the left subclavian artery, the aorta narrowed into a flat-topped mound at the top of which was a hole through which only the finest probe could be passed. The probe passed through a channel and, after overcoming a slight obstacle, into a portion of the descending aorta which measured 5 cm. in circumference at this point and showed no fatty intimal plaques. All the abdominal viscera and the extremities showed moderate passive congestion. Brawny induration with hemorrhagic infarcts on the right was found in the lungs. Microscopic sections of various levels of the aorta and its tributaries were stained with various preparations. The most valuable of these for demonstrating the chro-

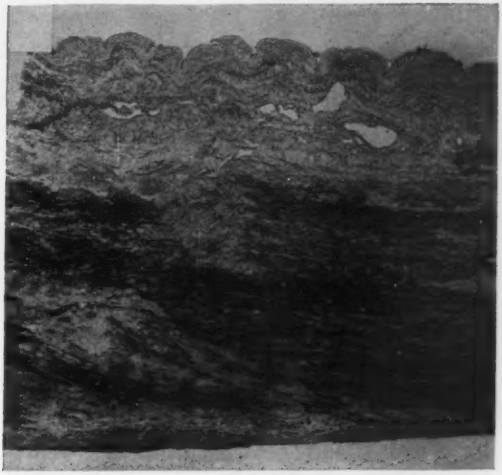


*Figure 1.—Aorta above stenosis illustrating media necrosis: elastic tissue stain (resorcinol fuchsin).*

motropic substance in the vessel wall was Feyrter's "closed staining" with tartaric acid thionine which brought out the red metachromasia of this substance in the vascular wall especially clear after previous treatment with 95 percent alcohol. The media of the descending aorta above the stenosis (fig. 1) showed the clearly demarcated elastic membranes which stained strongly with resorcinol fuchsin. Between the membranes and about the muscle fibers, especially the paler colored fibers, chromotropic substance was scantily deposited and was found mainly in the inner layers of the media. A slight thickening of the intima was seen through the heavy deposit of fine collagenous fibers. The changes in the transverse aorta beyond the tear were similar to those described. Interruptions were seen in the elastic membranes here and there and were in places visible only as fragments. The number of unstained muscle fibers, as shown by the lack of nuclei, was augmented in spots so that small areas of the media appeared empty of nuclei. In such places, the deposit of chromotropic substance was unusually heavy.

The elastic membranes of the ascending aorta near the tear (fig. 2) were interrupted in indefinitely bordered areas which sometimes ran perpendicular to the intima and sometimes parallel to it. Such areas might run into each other revealing wholly irregular, elastica-free regions. Where elastic tissue remained, the membranes did not run uniformly, but consisted of broken fragments of elastic tissue held together by fine fibers or more homogeneous masses which stained palely with resorcinol fuchsin. The regions of muscle fiber disappearance appeared more frequent and larger than in the transverse aorta and in such areas the fragmentation of the elastica appeared increased. Chromotropic substance was abundant and often appeared as a cystic collection. Here, there were also signs of growth of young connective tissue.

At the site of the tear the edge was a slightly rounded ridge covered by intima, and a projection of the media from the rupture site in the form of a flap appeared in only one section. At the border of the rupture the media ended almost entirely; only the outermost layers could be recognized as scattered, partially clumped elastic fibers and membranes at the base of the rupture. The inner

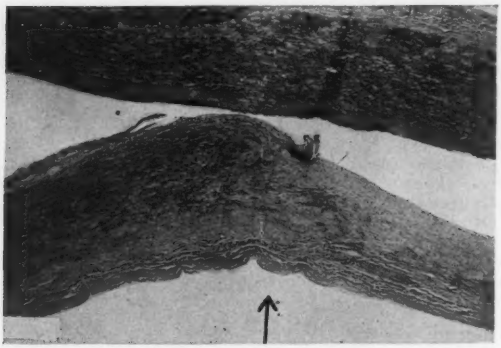


*Figure 2.—Aorta at site of rupture (arrow) and adjacent aortic elastic tissue (resorcinol fuchsin stain).*

layers of the ruptured area were formed by young connective tissue rich in star-shaped cells that also clothed the cliff like edge of the rupture and was continuous with the intima of the intact portion of the aorta. The adventitia contained more fibers and was more densely woven in the ruptured area. On examination the narrowed portion of the aorta in serial section revealed that the lumen was not completely obliterated, but was narrowed to between 1 and 2 mm.; the diameter of the whole aorta in this area was about 8 mm.; and the thickness of the media was 3 mm. The whole intima in this area was thickened by fibrosis. The media contained irregularly coursing elastic membranes and the media of the internal mammary, internal carotid, and subclavian arteries contained from 15 to 20 parallel coursing and communicating elastic membranes. Chromotropic substance was abundant, and the intima was thickened by extremely fine fibers and was about half the thickness of the media. The medial necrosis here was as severe as in the ascending aorta in contrast to the descending aorta which was only slightly involved.

The liver, spleen, and trachea showed only the signs of chronic, passive congestion. The pathologic diagnosis was (*a*) coarctation of the aorta with definite collateral vessel formation, (*b*) a bicuspid aortic valve, (*c*) medial necrosis of the aorta with a healed rupture, and (*d*) enlargement and dilatation of the heart with congestive failure.

## DISCUSSION

The coexistence of aortic aneurysm and coarctation of the aorta was discussed by Hamilton and Abbott (*6*) who collected 38 cases. Of these, 15 came to autopsy, 14 of which showed microscopic changes consisting of medial alterations interpreted then, as representing degenerative processes weakening the medial walls. These were all studied before the publication of Erdheim's paper on the specific changes of medial necrosis and now probably would be classified as such (*14*). Only two cases, both published subsequently, showed the changes of medial necrosis above the coarctation with normal aortas below (*1*) (*4*). In neither case was the histologic findings of the

*Figure 3.—Roentgenogram of excised sternum and anterior portion of the ribs illustrating anterior notching and erosion of the ribs and enlargement of the mammary arteries.*

other elastic-walled vessels mentioned. The microscopic findings of the vessels involved in the collateral circulation that arises in aortic coarctation is rarely mentioned in the literature. The present case is of interest as a third case in the literature in which there was medial necrosis above the coarctation and none below it. It is of special interest in that the medial necrosis extended into all other elastic

vessels which were subject to the patient's hypertension. This was even true of the internal mammary arteries which, in addition, showed an elastic type of structure instead of the usual muscular type. All of this suggests hypertension as a special factor in the etiology of medial necrosis.

Concerning the healed rupture the question arises as to whether this was really a rupture, perhaps 6 months old, in which intimal proliferation had covered a shallow tear, the media of which was simultaneously strengthened by a young connective tissue proliferation. Another possibility is that this represented a stage in every rupture caused by medial necrosis, a stage at which the media is stretching and yielding while the connective tissue formation is gradually falling behind in the process of repair, the intima, meanwhile, covering the weak stretching and widening chasm. In such a condition the strain would eventually become great enough to cause rupture of the few remaining bridging elements. The tear would then suddenly become complete and the typical findings of aortic rupture would ensue. This hypothesis could be checked by a microscopic review of fresh cases of rupture, looking for evidence of a previous connective tissue proliferation at the edges and base of the tear.

It is interesting that the coarctation was completely missed clinically, despite several fluoroscopic and roentgenographic examinations of the chest for heart size. In view of the prominent rib erosion noted on the roentgenogram of the removed anterior chest wall after death (fig. 3), it must be assumed that the changes were not found previously because they were not looked for. It is, therefore, important to reemphasize the fact that coarctation frequently does not cause death until late in life.

## REFERENCES

1. KENNEY, T. D.; SYLVESTER, R. E.; and LEVINE, S. A.: Coarctation and acute dissection of aorta associated with pregnancy. Am. J. M. Sc. **210**: 725–732, Dec. 1945.
2. STRASSMAN, G.: Ploetzlicher Tod bei Stenose des Isthmus Aortae. Beitr. z. gerichtl. Med. **5**: 91, 1922.
3. KATZ, H.: Ueber den ploetzlichen natuerlichen Tod in Schwangerschaft, Geburt und Wochenbett. Arch. f. Gynäk. **115**: 283–312, Nov. 1922.
4. HARRISON, F. F.: Coarctation of the aorta of the adult type associated with cystic degeneration of media in first portion of arch. Arch. Path. **27**: 742–747, April 1939.
5. GLOGGENGIESSER, W.: Sogenannte Spontan Rupture der Aorta bei Isthmusstenose und Media Necrosen der Aorta. Ztschr. f. Path. **56**: 11, 1941.
6. HAMILTON, W. F., and ABBOTT, M. E.: Coarctation of aorta of adult type. Am. Heart J. **3**: 381–421, April 1928.
7. ABBOTT, M. E.: Coarctation of aorta of adult type. Am. Heart J. **3**: 574–618, June 1928.

8. SCHNITKER, M. A., and BAYER, C. A.: Dissecting aneurysm of aorta in young individuals, particularly in association with pregnancy with report of case. Ann. Int. Med. **20:** 486–511, March 1944.
9. Handbuch der Speziellen Pathologischen Anatomie und Histologie. Volz Berlin, 1924.
10. BONNET, L. M.: Su la lesion tête stenose congenitale de l'aorte. Rev. de Med., Paris **23:** 108–481, 1903.
11. BLALOCK, A., and PARK, E. A.: Surgical treatment of experimental coarctation (atresia) of aorta. Am. Surg. **119:** 445–456, March 1944.
12. CRAFOORD, C., and NYLIN, G.: Congenital coarctation of aorta and its surgical treatment. J. Thoracic Surg. **14:** 347–361, Oct. 1945.
13. SHUMACKER, H. B., Jr.; Coarctation and aneurysm of aorta; case report treated by surgery. Ann. Surg. **127:** 655–665, April 1948.
14. ERDHEIM, J.: Medionecrosis aortae idiopathica. Virchow's Arch. f. Path. anat. **273:** 454, 1922; and **276:** 187, 1924.

## Pulmonary Resection for Lung Abscess[1]

A review of the literature reveals a case fatality rate of about 37.5 percent in hospitalized cases of pulmonary abscess. Transthoracic drainage and chemotherapeutic agents have greatly improved the progress in acute cases, but the mortality in chronic cases has remained high. A classification of lung abscess as simple and complicated is extremely useful. The simple abscess is characterized by a single cavity and no associated pathologic changes. The complicated abscess, on the other hand, may have several daughter abscesses, extreme pneumonitis, fibrosis, or branchiectasis. While adequate drainage of a simple lung abscess may result in cure, drainage of the major focus of a complicated abscess, with healing of that abscess cavity, still leaves significant pulmonary pathologic changes present, and the patient cannot be considered well.

The preoperative study of these patients should include X-ray localization of the cavity, bronchoscopy, and bronchography. At the Fitzsimons General Hospital, pulmonary resection was performed in a series of 38 cases, 36 of them complicated. Bronchiectasis was the most frequent complication. Pulmonary resection is the treatment of choice for complicated lung abscess. The management of upper lobe abscess presents a special problem because of inadequacy of bronchoscopic visualization of this area. Lung abscesses occurring in the upper lobes should be treated by pulmonary resection regardless of whether or not complicating factors can be demonstrated preoperatively. This is of particular importance in the management of patients in the middle and older age groups because of the possibility of the presence of a bronchogenic carcinoma. Postoperative empyema occurred in 5 patients in this series, and death occurred in 3, a fatality rate of only 7.9 percent.

[1] NEERKEN, A. J., and GROW, J. B.: Abstr. J. Thoracic Surg. 18: 738–741, Oct. 1949.

# Renal Infarction

## Report of Two Cases

URQUHART L. MEETER, *Major, MC, U. S. A.*[1]
JACK W. SCHWARTZ, *Colonel, MC, U. S. A.*[1]

THE subject of renal infarction was recently well-covered by Regan and Crabtree (*1*) who classified infarctions into 3 etiologic categories; venous, arterial, and traumatic. They recorded the incidence as follows: venous 20, arterial 47, and traumatic 3; and added 4 cases of arterial infarction. The apparent rarity of this entity may be attributed to unreported cases. Stegeman (*2*) added a case of traumatic thrombosis with infarction, complicated by an attempted surgical repair of a ruptured kidney with eventual nephrectomy. Shea, Schwartz, and Kobilak (*3*) in a review of the literature disclosed 10 cases of renal artery thrombosis not mentioned in the previous articles.

We desire to add four cases of massive arterial infarction of the kidney to the literature. Two of these have been previously reported under different considerations (*3*) (*4*). The first case was that of a Goldblatt phenomenon secondary to an organized thrombosis in the renal artery with apparent cure of the hypertension following nephrectomy. The second was an incidental discovery in a patient with a retrocaval ureter in a nonfunctioning kidney which was treated by nephrectomy.

### CASE REPORTS

*Case 1.*—A 19-year-old man sustained an injury to his left flank in an automobile accident on 8 December 1946, and was transferred to this hospital on 8 January 1947. He had had gross hematuria for 2 days following his accident but none since. Since childhood he had had intermittent attacks of acute radiating pain in the right flank usually of 1 or 2 days' duration. His only complaint on admission was a dull ache in the left lumbar region. Physical examination was negative except for moderate tenderness in the left flank and left lower quadrant of the abdomen. The urine on admission showed 1 to 2 plus albumin and 8 to 10 leukocytes per high-power field. Repeated blood pressure readings were normal. Intravenous urograms revealed a nonfunctioning left kidney and a congenital right hydronephrosis (fig. 1). No flow of urine was observed from the left ureteral orifice on cystoscopy. Both ureters were catheterized easily. Phenolsulfonphthalein appeared from the right catheter in 6 minutes and 12 percent was excreted in 10 minutes. None appeared from the left. The out-

[1] Letterman General Hospital, San Francisco, Calif.

lines of a normal upper tract on the left and a congenital hydronephrosis on the right secondary to a ureteropelvic obstruction was seen in the retrograde films (fig. 2). A Foley type pyeloplasty was performed on the right kidney on 11 February. The postoperative course was uneventful and, following a convalescent furlough, the left kidney was removed on 19 May. The left kidney was embedded in dense perinephric adhesions. The patient was returned to duty on 9 June.

*Pathologic report.*—The left kidney weighed 30 gm.; on section, very little normal kidney was noted grossly. Microscopically, an extensive necrotic process with ghostlike outlines of glomeruli and tubules were seen. In only one small area were functioning glomeruli found. Interstitial lymphocytic infiltration, shrunken tubules, and pink amorphous casts in the proximal convoluted tubules were described. The pathologic diagnosis was old traumatic infarction of the left kidney.

*Case 2.*—A 54-year-old man was admitted to this hospital on 7 January 1949 complaining of pain in both flanks and petechiae on his hands, feet, and sclerae. His past medical history revealed that he had had polycythemia vera for 3½ years and had been treated by repeated phlebotomies and with radioactive phosphorus. His present illness started on 19 December 1948 with chills, fever, and acute radiating pain in the left upper quadrant of the abdomen associated with gross hematuria. A tender mass appeared in the left upper abdominal quadrant but the pain subsided in 10 days. He then noted the petechiae for the first time. Physical examination showed a slightly tender mass in the left upper abdominal quadrant extending four fingerbreadths below the costal border and a boggy tender prostate. Blood pressure readings were normal. Blood counts showed 7.5 million erythrocytes; hemoglobin 15 gm. On repeated examinations the urine contained 1 to 4 plus albumin and many leukocytes; on several occasions erythrocytes and granular casts were found.

Intravenous pyelograms revealed a nonfunctioning left kidney and a normal right kidney. A large splenic shadow filled the left upper abdominal quadrant and extended to the iliac crest. Cystoscopic examination disclosed that clear spurts were seen from the right ureteral orifice with no flow from the left; both

*Figure 1.*

*Figure 2.*

*Figure 3.*

ureters were catheterized with ease. Normal excretion of phenolsulfonphthalein was obtained from the right catheter but no excretion from the left after 35 minutes. Retrograde pyelograms were normal (fig. 3). Retrograde pyelograms made 2 months later revealed no change. The patient continued to have mild attacks of pain in both flanks. Surgical intervention was contra-indicated because of the possibility of precipitating a fatal thrombosis of his right kidney.

## DISCUSSION

The diagnostic criteria for massive renal infarction caused by renal artery thrombosis are: (*a*) An onset with acute pain in the flank in which trauma may or may not be a factor; (*b*) a nonfunctioning kidney as revealed by intravenous pyelograms and divided excretion tests; (*c*) normal findings in the retrograde pyelogram; (*d*) a history of gross or microscopic hematuria and albuminuria; and (*e*) in some patients a cardiovascular disease or a blood dyscrasia. Although kidney infarction is mentioned in Cecil's Textbook of Medicine as a complication of polycythemia vera, a perusal of the literature fails to disclose any recorded cases. Case 2 was not proved by operation or autopsy, but it fulfills the previously mentioned criteria. The functionless infarcted kidney should be removed except when there is a definite contra-indication to operation. It definitely increases the chances for infection and the development of hypertension.

### REFERENCES

1. REGAN, F. C., and CRABTREE, E. G.: Renal infarction; clinical and possible surgical entity. J. Urol. 59: 981–1018, June 1948.
2. STEGEMAN, W.: Renal vessel thromboses. Urol. & Cutan. Rev. 52: 710–712, Dec. 1948.

3. SHEA, J. D.; SCHWARTZ, J. W.; and KOBILAK, R. W.: Thrombosis of left renal artery with hypertension; case report. J. Urol. 59: 302–306, Mar. 1948.
4. SCHWARTZ, J. W., and IREY, N. S.: Post-caval ureter; report of case. Urol. & Cutan. Rev. 53: 327–329, June 1949.

## Residency Training

To permit advanced planning and the careful selection of applicants, applications for residency training in military hospitals for any of the specialties may be forwarded at any time to the cognizant office of the Army, Navy, or Air Force:

*Army:* The Surgeon General, Attention Personnel Division, Career Management Branch.

*Navy:* Chief, Bureau of Medicine and Surgery, Attention Professional Division.

*Air Force:* The Surgeon General, U. S. Air Force, Attention AFCSG–26.

The final date for which applications will be considered for training to begin as of 1 July of any given year for Army and Air Force residents will be announced soon.

The Advisory Board of the Bureau of Medicine and Surgery will announce the beginning date for Navy residency training by letter.

Residencies in certain specialties such as General Surgery, Internal Medicine, Dermatology and Syphilology, and Obstetrics and Gynecology are limited, whereas they are readily available in other specialties, such as Pathology, Radiology, Urology, Psychiatry, Physical Medicine, and General Practice.

Application for residency training should be accompanied by the required service agreement, indicating the period of service to be given upon the completion of the training provided. Selection for residency training is contingent on acceptance of Regular Army, Navy, or Air Force commissions.

# Lower Nephron Nephrosis

## Report of Two Cases Treated With Intravenous Procaine

ALTON C. ABERNETHY, *Captain (MC) U. S. N.*[1]
ARTHUR J. WILETS, *Lieutenant (MC) U. S. N.*[1]
DAVID C. BEER, *Lieutenant (MC) U. S. N. R.*[1]

IN recent years there has been increasing interest in the methods of treatment of patients with lower nephron nephrosis. The authors recently noted an article by Friis (*1*) in which he suggested the use of intravenous procaine in the therapy of sulfathiazole anuria. It is believed that this method might be useful as an adjunct in the treatment of this disease, and it was used in two cases of oliguria brought on by different etiologic factors.

### CASE REPORTS

*Case 1.*—White man, age 72. At the time of admission his physical examination was essentially negative except for moderate homogenous enlargement of the prostate gland; 90 cc. of residual urine were obtained during the examination.

A preliminary prophylactic bilateral vasectomy was performed and subsequently a transurethral resection was done with removal of about 20 gm. of tissue. Moderate bleeding occurred during the course of the operation.

The patient's general physical condition during the first 24 hours postoperatively was satisfactory, but it was noticed that he had only passed a total of 150 cc. of urine during that period. Intravenous procaine therapy was instituted at that time, using a 1 percent solution. The result of the procaine therapy on the urinary output is illustrated in figure 1. With the administration of intravenous procaine, the following measures were instituted:

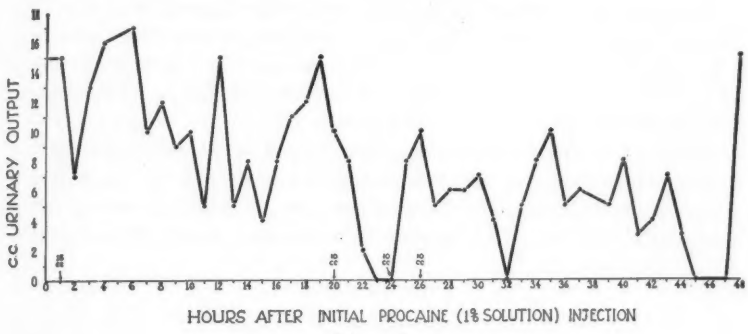



*Figure 1.*

[1] U. S. Naval Hospital, Oakland, Calif.

(*a*) Urine was collected in a jar connected to the indwelling catheter and measured accurately every hour.

(*b*) The patient received nothing by mouth.

(*c*) The fluid and caloric requirements were met by giving parenteral fluids to match the previous day's urinary output plus an amount equal to the loss by vomitus and by insensible means (thought to be about 1,000 cc. daily).

(*d*) Blood chemistry studies were done daily to determine the nonprotein nitrogen, carbon dioxide combining power, and blood chlorides. The changes in the blood chemistry values are illustrated in table 1.

TABLE 1

| Postoperative day | Nonprotein nitrogen | Carbon dioxide combining power | Blood chloride | Hemoglobin |
|---|---|---|---|---|
| | *Mg. per 100 cc. blood* | *Mg. per 100 cc. blood* | *Mg. per 100 cc. blood* | *Grams* |
| 1 | 66.4 | | | 12.5 |
| 2 | 82.6 | | | |
| 3 | 98.4 | 52 | | 9.0 |
| 4 | 94.8 | 46 | 410 | |
| 5 | 125.1 | 52 | 460 | |
| 6 | 128.3 | 37 | 420 | |
| 7 | 169.0 | 44 | 400 | 10.0 |
| 8 | 177.0 | 46 | 380 | 11.0 |
| 9 | 157.2 | 40 | 370 | |
| 10 | 142.0 | 44 | 390 | 11.0 |
| 11 | 217.0 | 38 | 400 | |
| 14 | 190.0 | | | |
| 15 | 184.0 | | | |
| 16 | | | 390 | |
| 21 | 124.0 | 68 | 470 | |
| 23 | 82.6 | 56 | 490 | |

(*e*) Acidosis was treated by the administration of one-sixth molar sodium lactate solution intravenously; the fall in blood chlorides was treated by administering normal saline with 5 or 10 percent dextrose as caloric requirements indicated.

The fluid intake, the total urinary output, and fluid loss by vomitus were charted over a period of 14 days: Charting was discontinued at this time, as it appeared that normal urinary output had been reached. The patient's general clinical appearance improved gradually during this period, paralleling his increase in urinary output. The nonprotein nitrogen was slow in returning to normal and 5 days after diuresis began the nonprotein nitrogen was still 82.6 mg. per 100 cc. blood.

He vomited daily, the amounts varying from 151 to 500 cc. during the first 8 days; thereafter, vomiting diminished. Oliguria (amounts varying from 97 to 643 cc.) persisted until the twelfth postoperative day at which time the urinary output increased to almost normal levels. From that time on until he died, he voided not less than 1,000 cc. in any 24-hour period. He was able to drink small quantities of fluid from the sixteenth to the twenty-seventh postoperative day. After that time frequent vomiting occurred and he required at least 3,000 cc. of intravenous fluids daily for supportive therapy. On the thirty-second postoperative day his blood pressure dropped to 90/50 and he was given an initial dose of 1.6 mg. digitoxin intramuscularly, then 0.2 mg. daily. This was of temporary aid and on the following day his blood pressure was 122/62. On the thirty-sixth day his general condition again appeared to be deteriorating. His respirations became rapid and shallow and he was placed in an oxygen tent. In spite of all supportive therapy he became worse and died, 37 days after

diuresis began. At autopsy, the primary cause of death was found to be hypostatic pneumonia.

*Case 2.*—A. M. J., a 25-year-old man, was admitted following injury in an automobile accident in which he sustained multiple fractures including a comminuted fracture of the pelvis and left femur, with concomitant massive edema. Deep shock which occurred immediately after the accident, was overcome with plasma and whole blood transfusions but on the fourth day extreme oliguria occurred. The previous day, the urinary output had been only 235 cc. When oliguria occurred, 15 cc. of 1 percent procaine was given intravenously. The urinary output during the next 24 hours decreased to a total of only 135 cc. In this case intravenous procaine was not repeated because of the lack of results from the initial dose. He was observed and treated in the same manner as case 1.

Ten days after oliguria appeared, his urinary output rose to 375 cc. On the eleventh day it was 637 cc. During this same period of time, his nonprotein nitrogen demonstrated a steady rise, reaching 200 mg. per 100 cc. blood on the eleventh day. His blood pressure also continued to rise, and on the eleventh day was 208/94. Although his fluid intake was restricted to 1,000 cc. plus an amount equal to the daily urinary output, his course was progressively downhill, and on the twelfth day after the onset of oliguria he had acute pulmonary edema. Supportive measures such as oxygen administered by positive pressure, digitoxin, and phlebotomy were used. He died approximately 6 hours after the onset of pulmonary edema. Autopsy showed the primary cause of death to be acute pulmonary edema.

## ETIOLOGY

Lower nephron nephrosis can be caused by a large number of conditions, including crushing injuries, sulfonamide intoxication, blood transfusion reactions, and intravascular hemolysis following transurethral prostatic resections.

Trueta (*3*) and his coworkers have found that, although the efferent vessel of the cortical glomerulus is of small caliber, the similar vessel of the juxtamedullary glomerulus is large, at times being equal to or even larger than the afferent vessel (fig. 2). They also found that the efferent vessels of the cortex drain into cortical capillaries of small caliber, while the efferent vessels of the juxtamedullary glomerulus drain into the vasa recta, which are straight medullary vessels of large caliber. The basic importance of the large caliber of these efferent vessels becomes apparent when one considers the large amount of blood that must flow through the juxtamedullary glomeruli when the medullary bypass acts as the principal vascular functioning unit.

Trueta has pointed out that in the presence of vasoconstriction, the blood will flow through the point of least resistance, i. e., through the large efferent vessels of the juxtamedullary glomeruli, rather than the comparatively narrower ones of the cortex. Also, it was demonstrated that the portions of the nephron receiving blood richest in oxygen are those situated in the medullary ray, as the capillaries surrounding

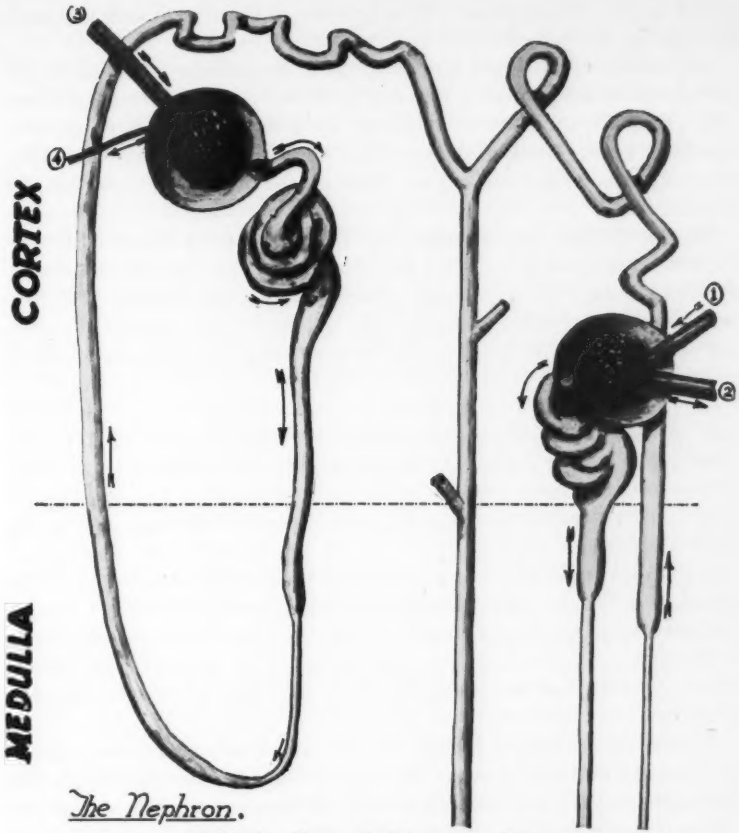

CORTEX
MEDULLA
The Nephron.
Note the relative equality in size of the afferent ① and efferent vessels ② of the juxtamedullary glomerulus as compared to that of the cortical glomerus ③④ (AS SUGGESTED BY TRUETA, et al STUDIES OF THE RENAL CIRCULATION)

*Figure 2.*

these portions obtain their blood supply directly from the efferent vessels of the cortical glomeruli. The proximal and distal convoluted tubules receive poorly oxygenated blood, as their capillaries receive blood that has already passed through the arterial side of the cortical intertubular bed. It is apparent that, in the event of a medullary bypass far less blood than normal would go to the renal cortex and the amount of oxygenated blood reaching the distal convoluted tubules

would be less than normal. This, in part, is the reason that much of the damage is confined to the lower nephron.

According to Milliken and Karr (*4*), the kidney is supplied by nerves which are primarily derived from the renal plexus. The renal plexus is made up of nerve fibers and ganglia, arranged in a network about the renal vessels and these fibers enter the kidney together with renal vessels, and then follow them to the finest divisions of the arterial tree.

In the opinion of Corcoran and Page (*7*), posttraumatic anuria (crush syndrome) is merely a special instance "of a group of related states of renal injury" falling in two patterns (*a*) a group in which primary lesion is an obstruction of the tubules by pigmented casts; and (*b*) a group in which injury is primarily necrosis of the distal convoluted tubules.

They believe that the underlying mechanism is a renal ischemia due to vasoconstriction. The ischemia causes oliguria and in the presence of aciduria a precipitation of pigmented casts occur. Myoglobinuria and hemoglobinuria, in association with renal ischemia, may result in severe renal injury; under normal conditions they would have little effect.

Corcoran and Page (*7*) also believe it is likely that in the crush syndrome "the humoral vasoconstrictors of severe shock act on the renal circulation in a manner similar to that of intense vasomotor stimulation, i. e., by causing constriction of the cortical interlobular arteries, with cortical ischemia and diversion of blood into the medulla."

Thorn (*6*) believes that oliguria and anuria may follow the reduced glomerular filtration secondary to renal vasoconstriction, shock, and decreased plasma volume. He states that later the oliguria and anuria be maintained by the increase in the intrarenal pressure, tubular obstruction, and a leakage of the glomerular filtrate through damaged tubular walls.

An important contributing factor is a decrease in effective filtration pressure consequent to the increased intrarenal pressure. The intrarenal pressure may be increased by dilatation of tubules, by interstitial edema, and by inflammatory exudate. Dilatation of a part of the tubules may be caused by obstruction either at or below the areas of dilatation, and the obstructing substances may be detritus of the tubular epithelial cells or pigment material.

Creevy (*13*) has pointed out that "Hemolysis can be seen to occur within the bladder during transurethral prostatic resections as soon as the irrigating water comes in contact with the blood; if large veins are opened, it is evident that water may enter the venous system and cause much more extensive hemolysis." He believes that stagnation of blood in the pelvic veins may increase the severity of the hemolysis and adds that one would consider it logical that hemolysis would be

more prone to occur when brisk bleeding would require the use of the irrigating fluid under a higher pressure than is normally used.

During the process of transurethral resection of the prostate, Foley (*14*) observed jets of blood coming from the ureteral orifices and immediately after this he discovered a high level of free hemoglobin in the plasma.

Landsteiner and Finch (*15*), investigating the possibility that distilled water used during transurethral resections of the prostate might gain access to the blood stream, added 1 gm. of sodium salicylate to each liter of irrigating fluid. They chose this substance, because of the ability to accurately determine its presence even in small amounts in the blood (*16*). They conclude from their observations, that about 500 cc. or more of distilled water is necessary to produce hemoglobinuria.

Creevy (*17*), in using 4 percent glucose solution as an irrigating fluid, measured the blood sugar before and after performing transurethral prostatic resections in 78 patients. He found that the average blood sugar preoperatively was 81 mg. per 100 cc. blood. Postoperatively the average level was 198 mg. per 100 cc. Twenty-four of the patients had postoperative levels which exceeded 200 mg. per 100 cc. He concluded from this data that there was no question about the irrigating fluid entering the circulation in a high proportion of cases.

Fischer (*5*) believes that the capsular nerves are independent of the nerves found in the renal parenchyma and has described a network of capsular nerves which start at the kidney hilum. He also found that stimulation of the splanchnic trunk (which consists primarily of vasoconstrictors) decreased the renal blood flow as well as the quantity of urine secreted. On the other hand, a secretion of this same splanchnic trunk resulted in both an increase in renal blood flow and urinary secretion.

Mason and Mann (*8*) have found that hemolysis will cause a reduction of renal blood flow because of local renal vascular spasm.

Flink (*9*) believes that free hemoglobin is a threshold substance appearing in the urine in plasma concentrations exceeding 135 to 290 mg. per 100 cc. This free hemoglobin disappears from the circulating plasma in approximately 36 hours; on reaching the kidneys it produces vasoconstriction.

Yuille et al. (*10*) have demonstrated in experimental animals, that tubular necrosis follows anoxia and will result in extensive precipitation of hemoglobin in the tubules.

Peters (*11*) believes that the primary factor making possible a significant rise in intrarenal pressure is the relative rigidity and inelasticity of the renal capsule in the adult human being.

## SYMPTOMS

The chief symptoms and signs of the syndrome of lower nephron nephrosis are: (*a*) Shock; (*b*) vomiting; (*c*) oliguria and anuria; (*d*) hypertension (Lucke (*2*) found in his series of cases, that on the first day, there would be a drop in blood pressure to shock levels, on the second day there would be a return to normal, and on the third day he found a rise to approximately 150/90, following this there would be a maintenance at approximately this same level, or there would be a further increase); (*e*) edema (Lucke found this to be a variable phenomenon, usually being slight or moderate in extent); and (*f*) uremia with its attending symptoms and signs.

## PATHOLOGY

Lucke (*2*) has described in detail the pathologic changes found in the kidney in lower nephron nephrosis. He finds that the essential changes appear to be restricted to the lower segments of the nephrons. Degeneration or actual necrosis involves the thick tubules of Henle as well as the distal convoluted tubules. Edema and cellular reaction occur in the stroma around the more severely damaged portions of the tubules. Thrombosis of adjacent veins in this area is frequently a concomitant finding. He found casts of a heme compound lying within the lumens of a variable number of tubules in the lower segments and of portions of the nephrons (fig. 3), i. e., the renal corpuscles and the proximal and intermediate segments.

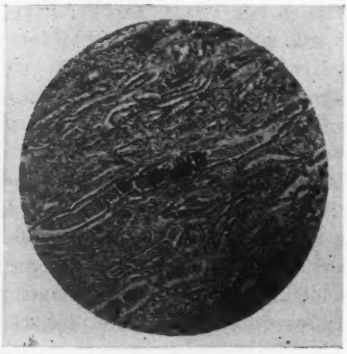


*Figure 3.—Photomicrograph of postmortem kidney tissue (× 200). Note deposition of pigment in tubules even though the patient died 37 days after the onset of oliguria.*

Ayer and Gauld (*18*) have described the sequence of pathologic changes in the kidneys following intravascular hemolysis. They present the autopsy findings in 7 patients who died 3 hours to 10 days following hemolytic reactions:

*Three hours after hemolysis:*

(*a*) Glomerular swelling.

(*b*) Slight cloudy swelling of the proximal convoluted tubules and pale acidophilic material in their lumens, becoming dark red to brown in the distal convoluted and collecting tubules.

*Third day:*

(*a*) Considerable brick-red pigment within the cells of the distal convoluted tubules and some sloughing of these cells.

*Sixth day:*

(*a*) Considerable interstitial edema and leukocytic infiltration around the convoluted tubules.
(*b*) Marked desquamation of the tubular cells.
(*c*) Some of the pigment of the lumens appeared to be turning greenish-yellow.

*Seventh day:*

(*a*) A few mitotic figures began to appear in some of the tubular cells, which the authors considered an indication of attempts at repair.
(*b*) The destruction of tubular cells as well as interstitial infiltration and edema were still pronounced.

*Tenth day:*

(*a*) There now appeared complete destruction of some of the tubules, these being replaced by inflammatory cells and fibroblasts.
(*b*) Numerous cellular casts were present in the distal convoluted and collecting tubules.
(*c*) Nearly all the inflammation appeared to be around the distal convoluted tubules.

Some have claimed that the deposition of casts is the primary cause of anuria. Lucke (*2*) has found a large discrepancy in the number of casts found in different cases. He states that they are rarely entirely absent, although in some cases, these casts are conspicuous in every microscopic field.

The regeneration of the tubular epithelium is rapid; Lucke found that within less than 10 days the majority of the damaged areas are completely relined.

McLaughlin et al. (*19*) believe that one can usually explain the anemia associated with the oliguria syndrome on the basis of hemolysis, hemorrhage, hemodilution (as that found with irrigating solution), and a depression of hemopoiesis by nitrogenous wastes.

Trueta et al. (*3*) have raised the question as to the possibility that a diversion of the intrarenal blood through the medullary bypass could cause a cortical ischemia of sufficient degree to cause the liberation of a pressor substance, thus causing hypertension.

## TREATMENT

It has been demonstrated (*12*) that stimulation of the renal vasomotor nerves diverts the renal blood flow from the cortex (which ordinarily receives most of the blood flow) to the medulla, resulting in renal cortical ischemia. On the basis of experimental work it appears that this vasomotor mechanism may be the basis for the renal cortical ischemia seen in the crush syndrome and similar conditions, and that

renal injury, as a result of this mechanism, may be prevented by blocking the renal vasomotor pathways by high spinal anesthesia and/or splanchnic block.

Corcoran and Page (*7*) think that clinical procedures which block the renal nerves should be used in the early treatment of the crush syndrome as well as the related conditions. No value is found in this type of therapy once the damage from renal ischemia, pigmentary occlusion, and toxic injury has been established.

Thorn (*6*), in discussing lower nephron nephrosis "with intact and relatively uninvolved glomeruli," suggested the use of whole blood to restore a normal circulating blood volume during the period of shock, the restriction of fluids to approximately 1,000 to 1,500 cc. per 24-hour period, in addition to the amount lost by fever, vomiting, and so forth. He advises as high a caloric intake as possible, the addition of small amounts of plasma or serum albumin to the *d*-glucose solution, and the use of digitalis at the first indication of pulmonary congestion or cardiac enlargement. He emphasized that the hypochloremia frequently appears after diuresis has started and warns of the frequent necessity of administering large quantities of sodium chloride.

Friis (*1*) has recently suggested a somewhat different approach to the treatment of certain types of anuria. He administered intravenous procaine to a patient with sulfathiazole anuria and was able to observe diuresis very soon afterwards. He believed that this was probably caused by a transient blocking of the sympathetic nerves, "perhaps with direct action on the renal functions." This method was considered to be of some value, particularly in view of Trueta's (*3*) recent work, and that by this means one might be able either to preclude or combat the medullary bypass.

Snapper (*20*) believes that the daily fluid intake should not exceed 800 cc. with additional fluid being used only to replace that lost by vomiting, diarrhea, and so forth. The electrolyte and protein intake should also be reduced. He discussed the diet advocated by Borst (*21*), who suggested 200 gm. of butter and 200 gm. of sugar daily. This is obviously of high caloric content, but, as Snapper pointed out, is very unpalatable.

In those cases of anuria, caused by intravascular hemolysis, some authors (*22*) (*7*) believe that immediate alkalinization of the urine is indicated despite the conflicting experiments concerning this procedure.

It has been said that

> The anuric patient differs from other patients in an important respect: Water and not salt is the important factor in edema formation. Without renal excretion there is a fixed daily fluid output through other channels. If the fluid intake exceeds this, edema follows, regardless of the electrolyte * * *. With

its volume limited by a fibrous capsule, the kidney parenchyma is * * * affected by edema (*22*).

These authors also believe that if cardiac function is impaired at the onset of anuria, one may anticipate that the patient will not survive the critical period of 1 or 2 weeks.

Methods other than purely medical ones, in the treatment of anuria have numerous proponents. Some of the more popular are: (*a*) peritoneal lavage; (*b*) the artificial kidney; (*c*) intestinal irrigation; and (*d*) exsanguinotransfusion.

Snapper (*20*), in a recent article, gives an excellent review of these procedures. He believes that, in lower nephrosis, the regeneration of the tubules always starts after 10 days, and that treatment must be directed to assure the survival of the patient for the first 10 to 14 days following the onset of anuria. The reduction in the intake of fluid, electrolytes, and proteins is the prime consideration in most cases of acute anuria, and in most cases is all that is required. Occasionally, it may be necessary to use the other procedures noted. Exsanguinotransfusion has a number of handicaps, including a laborious procedure, and the expense of a necessarily large amount of blood. Intestinal irrigation carries the danger of serious changes in the electrolyte content of the blood. He believes that peritoneal dialysis and the use of the artificial kidney appear to be the more satisfactory of the procedures.

Ormond and Best (*23*), however, believe that gastric and duodenal lavage is both satisfactory and simple to perform.

Lattimer (*24*) reports a regimen in which conservative methods would ordinarily be employed for a period of about 14 days. If diuresis had not started by this time, bilateral renal decapsulation would be attempted.

Reif and Bramley (*25*) reported a patient with mercury poisoning, in which unilateral decapsulation was performed 3 days after poisoning. They found that urine formation was apparent soon thereafter and concluded that the function of the decapsulated kidney was superior to the other one for many weeks after the procedure.

Vermeulen and Snead (*26*) did considerable experimental work with dogs, which they poisoned with mercuric chloride and then performed decapsulations. They concluded that their experiment demonstrated no support for the use of decapsulation in uremia due to mercury poisoning.

Kleiman (*27*) reports a case of sulfathiazole anuria with a prompt response to renal decapsulation. He believes that patients with sulfonamide anuria should first be given symptomatic therapy including ureteral catheterization. If these measures fail, however, renal decapsulation (preferably bilateral) should be performed. He thinks that

this is especially indicated in those cases in which sulfonamide crystals are not recovered from ureteral catheterization. He adds that "No patient suffering from anuria caused by sulfa drug sensitivity should be permitted to die from anuria without surgical intervention (decapsulation), unless there are hopeless organic conditions which contraindicate the operative procedure."

Abeshouse and Tankin (*28*) believe that decapsulation is indicated in those cases of persistent oliguria or anuria which follow sulfadiazine or sulfathiazole administration after the presence of calculi or concretions in the pelvis or ureter have been ruled out. In the case of nonobstructive anuria of the reflex or secretory type, the authors believe that one can trace the beneficial effect of decapsulation directly to the interruption and destruction of the sympathetic nerve supply to the kidney.

Shapiro (*29*) urges early decapsulation in certain types of sulfonamide anuria, warning of the danger of irreversible kidney changes that may occur if the anuria persists too long.

A number of authors (*30*) (*31*) (*32*) have reported cases of oliguria and anuria following transfusion in which they attributed beneficial results to renal decapsulation.

## SUMMARY

Lower nephron nephrosis occurs after such conditions as crushing injury, sulfonamide intoxication, blood transfusion reaction, intravascular hemolysis, and following transurethral prostatic resection. There are a number of concepts regarding the mechanism of anuria in lower nephron nephrosis. The work of Trueta and others indicates that the underlying factor is a renal cortical ischemia which occurs because of stimulation of the renal vasomotor nerves and diversion of the renal blood flow from the cortex to the medulla. The cortex becomes ischemic and thus occurs the renal cortical injury seen in the crush syndrome and allied states. It has been suggested that this mechanism may be prevented by blocking the renal vasomotor pathways, e. g., caudal high spinal anesthesia or splanchnic block. As Corcoran and Page have suggested, this is well worth trying early in these conditions.

In the cases presented, no increase in diuresis followed the intravenous injection of procaine hydrochloride. Nevertheless, this still may be a step in the right direction to combat the deleterious effect of the medullary bypass on the function of the kidney. It may be that, in order to effectively accomplish this, the amount of procaine administered will have to be increased. Perhaps, nerve block or decapsulation with denervation will prove to be the most effective

single procedure (other than dietary and fluid restriction) in the treatment of lower nephron nephrosis.

Treatment should include restriction of fluid intake to an amount equal to that lost; maintaining protein electrolyte and *p*H balance. If the patient survives for a period of 10 to 14 days, regeneration of the tubule will occur usually after the tenth day and diuresis will then begin.

## References


1. Friis, N. P.: Sulfathiazole anuria cured by means of intravenous procaine treatment. J. Urol. **61**: 184–186, Feb. 1949.
2. Lucke, B.: Lower nephron nephrosis. Mil. Surgeon. **99**: 371–396, Nov. 1946.
3. Trueta, J.; Barclay, A. E.; Daniel, P. M.; Franklin, K. J.; and Pritchard, M. M. L.: Studies of the Renal Circulation. Charles C Thomas, Springfield, Ill., 1947.
4. Milliken, L., and Karr, W. G.: Influence of nerve on kidney function in relation to problem of renal sympathectomy. J. Urol. **13**: 1–23, Jan. 1925.
5. Fischer, K.: Anatomie und physiologie der nervi proprii der nierien kapsiel und ihre bedeutung fur die nierien chirurgie. Deutsch. Ztschr. f. chir. **222**: 228–273, Feb. 1930.
6. Thorn, G. W.: Treatment of renal insufficiency. J. Urol. **59**: 119–148, Feb. 1948.
7. Corcoran, A. C., and Page, I. H.: Crush syndrome. J. A. M. A. **134**: 436–441, May 31, 1947.
8. Mason, J. B., and Mann, F. C.: Effect of hemoglobin on volume of kidney. Am. J. Physiol. **98**: 181–185, Sept. 1931.
9. Flink, E. B.: Certain aspects of hemoglobinemia: Thesis of the Graduate School of Medicine of the University of Minnesota, 1945.
10. Yuille, C. L.; Gold, M. A.; and Hinds, E. G.; Hemoglobin precipitation in renal tubules: study of its causes and effects. J. Exper. Med. **82**: 361–374, Nov. 1945.
11. Peters, J. T.: Oliguria and anuria due to increased intrarenal pressure. Ann. Int. Med. **23**: 221–236, Aug. 1945.
12. Franklin, K. J.; Barclay, A. E.; Daniel, P.; Trueta, J.; and Pritchard, M. M. L.: Renal pathology in light of recent neurovascular studies. Lancet **2**: 237–238, Aug. 17, 1946.
13. Creevy, C. D.: Hemolytic reactions during transurethral prostatic surgery. J. Urol. **58**: 125–131, Aug. 1947.
14. Foley, F. E. B.: Personal communication to C. D. Creevy.
15. Landsteiner, D. K., and Finch, C. A.: Hemoglobinemia accompanying transurethral resection of prostate. New England J. Med. **237**: 310–314, Aug. 28, 1947.
16. Brodie, B. B.; Udenfried, S.; and Coburn, A. F.: Determination of salicylic acid in plasma. J. Pharmacol. & Exper. Therap. **80**: 114–117, Jan. 1944.
17. Creevy, C. D.: Importance of hemolysis during transurethral prostatic resection; a clinical investigation. J. Urol. **59**: 1217–1232, June 1948.
18. Ayer, G. D., and Gauld, A. G.: Uremia following blood transfusion; nature and significance of renal changes. Arch. Path. **33**: 513–532, April 1942.
19. McLaughlin, W. L.; Holyoke, J. B.; and Bowler, J. P.: Oliguria following transurethral resection of prostate gland. J. Urol. **58**: 47–60, July 1947.
20. Snapper, I.: Management of acute renal failure. Bull. New York Acad. Med. **25**: 199–227, April 1949.

21. BORST, J. G. G.: Protein catabolism in uremia. Lancet 1: 824–828, May 29, 1948.
22. BURWELL, E. L.; KINNEY, T. D.; and FINCH, C. A.: Renal damage following intravascular hemolysis. New England J. Med. 237: 657–665, Oct. 30, 1947.
23. ORMOND, J. K., and BEST, J. W.: Anuria; report of 27 cases. J. Urol. 61: 608–623, March 1949.
24. LATTIMER, J. K.: Plan for management of anuria. J. Urol. 54: 312–317, Sept. 1945.
25. REIF, H. A., and BRAMLEY, H. F.: Bichloride of mercury poisoning. J. Urol. 49: 583–589, April 1943.
26. VERMEULEN, C. W., and SNEAD, C. R.: Effect of renal decapsulation in experimental mercury nephrosis. J. Urol. 60: 216–220, Aug. 1948.
27. KLEIMAN, A. H.: Sulfathiazole anuria with recovery following renal decapsulation. J. Urol. 56: 598–601, Nov. 1946.
28. ABESHOUSE, B. S., and TANKIN, L. H.: Renal complications of sulfonamide therapy. J. Urol. 56: 658–687, Dec. 1946.
29. SHAPIRO, I. J.: Decapsulation of kidney for anuria due to sulfathiazole. J. Urol. 59: 538–543, April 1948.
30. BANCROFT, F. W.: Anuria following transfusion, effect of decapsulation of both kidneys. Ann. Surg. 81: 733–738, April 1925.
31. RAVICH, A.: Transfusion reaction with anuria and uremia cured by unilateral renal decapsulation. M. Rec. 154: 403–405, Dec. 3, 1941.
32. FLO, S. D., and CUMMINGS, H. W.: Unilateral decapsulation of kidney for transfusion of oliguria. Surgery 14: 216–222, Aug. 1943.

## Surgical Management of Ulcer [1]

Surgical management of ulcer in the past has been far too empiric. A three-quarter gastric resection performed with due regard to established physiologic principles appears to thwart the obstinate ulcer diatheses which have remained refractory to psychiatric and medical management, more effectively than any known agency. The surgeon who relies upon the excision principle but who fails to excise enough stomach, or who leaves the antral mucosa or who re-establishes continuity by employment of a long proximal duodenojejunal loop—the surgeon who does any of these—is performing an ulcer-abetting operation and is inviting ulcer recurrence. The role of vagotomy in the management of ulcer remains to be determined.

[1] WANGENSTEEN, O. H.: Role of surgeon in management of peptic ulcer. Wisconsin M. J. 48: 915–919, Oct. 1949.

# Disfiguring Malocclusion

## Correction by Cosmetic Prosthesis

PIERRE O. EVANS, *Major, DC, U. S. A.*[1]

ON 30 August 1948, a 26-year-old woman reported to the dental clinic complaining of a disfiguring malocclusion (fig. 1). She was able to close her lips only by pursing them. The central incisors were becoming sensitive to air thermal changes, and there was no incising contact obtainable even with the furthermost protrusion of the mandible. She also had difficulty enunciating words containing "s," "sh," "ch," and "f." Roentgenograms and study models were obtained. The overjet measured 10.5 mm. and the lower anterior teeth were in slight linguoversion from the left cuspid to the right lateral incisor. The roots and teeth were of slightly larger size than would normally be expected. The alveolar bone was of good density and a mild general gingivitis was present. The profile was such that correction of the lower anterior teeth would accomplish little cosmetically, while the upper lip was foreshortened and curled.

*Figure 1.*

In view of these facts, it was decided that removal of the upper central and lateral teeth, and an alveolectomy, would give a satisfactory anterior maxillary reduction with a good cosmetic result, improved function, and better speech. Removal of the upper left cuspid in order to allow a more perfect arch alinement was considered, but because of the comparative strengths of the cuspid and first bicuspid as abutments this was not done. Trial removal of the teeth and an alveolectomy was performed on the model, and the amount of alveolectomy was determined to be between 4 and 5 mm. at the crest of the central region. On this basis, articulated models were prepared and a claspless partial denture constructed, as well as a clear acrylic surgical tray for accuracy in the alveolectomy.

The teeth and alveolar bone were removed by the oral surgeon under procaine anesthesia, using extraoral infra-orbital injections to reduce the chance of postoperative swelling. The partial denture was inserted and was worn for 3 months (fig. 2). The cuspids were rather long and since the terminal end of the root canal was about 4 mm. from the incisal edge, it was decided that 2 mm. reduction of that edge could be accomplished in the three-quarter crown preparations. Crowns were prepared on this basis, seated, and full impressions taken which were poured in artificial stone. A piece of retention mesh wax was

[1] Madigan General Hospital, Tacoma, Wash.

*Figure 2.*

*Figure 3.*

bent lengthwise into a V or narrow trough and placed along the crest of the ridge from the mesial surface of one cuspid crown to the other with the open portion of the trough tissueward. The labial portion was placed sufficiently toward the lingual surface to allow 1.5 to 2 mm. of tooth material to be placed in position to make the final labial arch form. The mesh was cast in three-quarter crown gold and spot soldered to the abutments which left the stone cast intact.

Teeth of the proper shape were chosen and the lingual surface of these teeth ground out to allow them to be set tightly against the gold mesh and make the final labial arch form. White beeswax was poured from the lingual aspect through the mesh and then carved into lingual tooth surfaces. The case was invested. the wax boiled out, and acrylic dough of the proper shade was forced through the mesh to the acrylic teeth and processed. The finished bridge showed gold only around the three-quarter crowns. The arch of the bridge was structurally strong because of the cast gold mesh. The attachment of the acrylic teeth to the mesh with acrylic was quite strong, and the compatibility of the gingival tissue to the acrylic material was excellent. The final cosmetic result is shown in figure 3, 3 months after the final bridge insertion.

# Cleft Palate Prosthesis

ELLSWORTH K. KELLY, *Lieutenant Colonel DC, U. S. A.*[1]

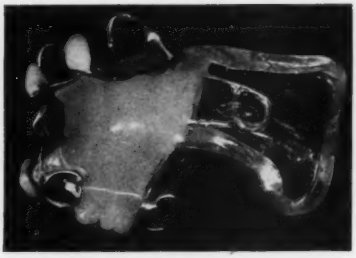
THE use of artificial material for closing the congenital cleft palate to aid speech and deglutition, dates from ancient times and probably was the first type of oral prosthesis. Many types and forms of obturators have been used in the past, but the restorations now used may be classified as the hinged bulb, the rigidly attached bulb, and the flexible velum. These are incorporated as part of a full or partial denture; or, if no teeth are missing, they are supported by a framework that clasps the natural teeth. While the flexible velum is theoretically the best and the patient easily learns to correct his speech with it, it fails because no flexible material is available that will not become foul in the mouth. Soft rubber and the more modern flexible plastics failed. The hinged bulb is used successfully by some dentists (*1*). The objections to it are that it allows escape of air, its construction is difficult, and it is easily broken. When methyl methacrylate resin and the chromium cobalt alloys became available, the solidly attached bulb came into general use. A bulb can be made from either of these materials: Both are relatively lightweight compared to the previously available vulcanite and gold. If it is desirable that the weight be reduced still further a hollow bulb can be constructed from either methyl methacrylate or the chromium cobalt alloys (*2*).

Fitz-Gibbon (*3*) has classified the various types of cleft palate as involving (*a*) the soft palate only, (*b*) the hard and soft palate, (*c*) the hard and soft palate and extending forward through the alveolar ridge and causing a single harelip, and (*d*) the hard and soft palate and extending through the ridge of each side of the premaxilla causing a double harelip. The first two conditions are most commonly found in the youth and young adult. If the defect was associated with harelip, the lip almost always has been previously repaired.

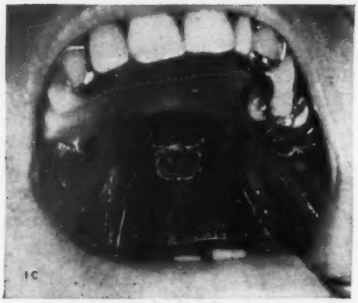
The prosthodontist will obtain the best results in a patient who has not had surgical interference in an attempt to repair the cleft. For this reason it is important that early cases are given thorough study and consultation to determine the treatment of choice. Surgical at-

[1] Valley Forge General Hospital, Phoenixville, Pa.

A

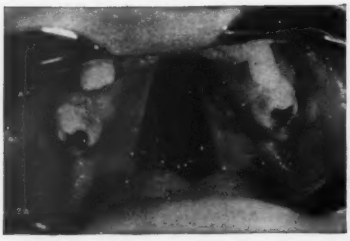


B

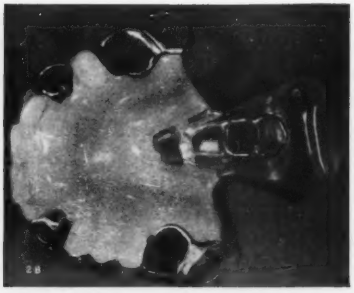


C

*Figure 1.*—A, *Wide cleft involving soft and hard palate almost as far forward as incisor teeth. Surgical closure had never been attempted. The patient had worn an inadequate device attached to a partial denture. Note crowns and inlays placed to afford adequate occlusal rests.* B, *Appliance for patient shown in* A. C, *Appliance in place.*

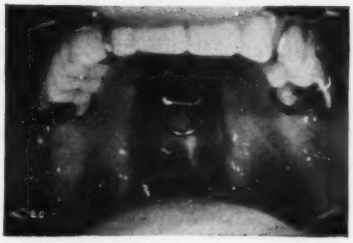


A

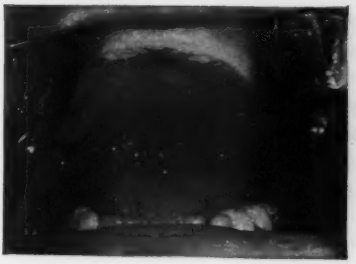


B

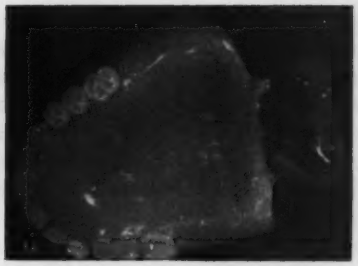
*Figure 2.*—A, *Cleft involving soft palate and hard palate up to height of the vault, a narrower cleft than that shown in figure 1. Surgical closure had never been attempted and the patient had worn a partial denture but not an obturator.* B, *Appliance for patient shown in* A. C, *Appliance in place.*



C



A

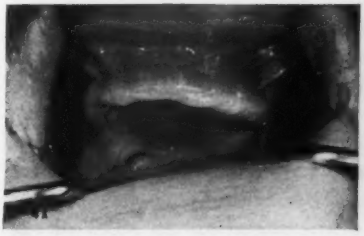


B

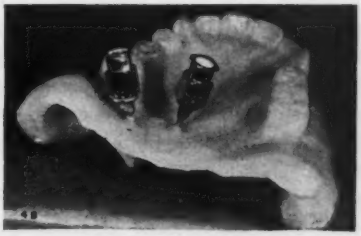
C

*Figure 3.—A, Edentulous maxillas with cleft involving soft palate only. The patient had worn a hinge-type obturator that had outlived its usefulness. Satisfactory retention was obtained with the intact hard palate and a solid type obturator proved highly successful. B, Appliance for patient shown in A. C, Appliance in place.*

tempts at closure when insufficient tissue is available to make a satisfactory repair, often result in a partially closed cleft with scar tissue and loss of tissue tone making further closure with a prosthesis impracticable. On the other hand, if the cleft is amenable to repair, surgical correction is far more satisfactory to the patient than prosthetic correction. In a few special clinics where the services of surgeons, orthodontists, prosthodontists, and speech specialists are available, the treatment is determined after consultation and study of the patient



A



B

*Figure 4.—A, Small perforations of hard palate following gunshot wound. B, Stainless steel hypodermic needles incorporated in the denture so that air can be exchanged between oral and nasal cavities without dislodging it.*

869434°—50——5

by all concerned. At such clinics the patient gets the treatment most suitable for his condition; treatment is not determined by his fear of surgery, his economic status, or a surgeon's view that operation is the only treatment regardless of the prognosis.

In military practice, despite physical requirements for enlistment, we occasionally encounter a patient who needs prosthetic restoration of a congenital cleft palate. Dependents of military personnel with cleft palate may also be seen.

The following describes the manufacture of a solid obturator attached rigidly to the partial or full denture after the manner of Fitz-Gibbon (*3*) and Bucknam (*4*). Such an obturator is the simplest and most durable and it will give satisfactory results in closing the cleft. The obturator must have a rigid and highly retentive denture for attachment. The denture, if it is partial, should be well supported with adequate occlusal rests. If anterior teeth (cuspids or incisors) must be used for abutments, three-quarter crowns or inlays must be placed to afford deep rests for the clasps which direct the stress in the direction of the long-axis of these teeth. Three-quarter crowns were used for this purpose in the patient shown in figure 1, and inlays were used to afford positive rests in the cuspids in the patient shown in figure 2. The edentulous patient is the most difficult to treat. Since the patient shown in figure 3 had a cleft of the soft palate only, an adequate posterior seal could be obtained and retention was no problem. Figure 4 illustrates an acquired cleft following a gunshot wound and is included to show how retention can be obtained in a patient with a large opening in the hard palate but with a patent soft palate. Many congenital cleft palates present a similar problem when repair is attempted, and a closure of the posterior portion of the cleft leaves a large opening in the hard palate. A 19-gage stainless steel hypodermic needle was incorporated in the acrylic during processing so that its lumen formed a small communication between the oral and nasal cavities. In this way the denture retained its peripheral seal and since the obturator formed a seal around the perforation, the denture had good retention. Otherwise the difference in air pressure between the nasal and oral cavities created when the patient smokes, swallows, blows his nose, or coughs, will dislodge the denture no matter how well it is adapted.

The soft palate remaining on each side of the congenital cleft is usually movable and easily displaced, which makes impracticable the construction of the obturator on the model at the same time that the denture is processed. Furthermore, the shape of the sides of the obturator and the posterior border must be determined by the movements of the palatal and pharyngeal muscles. For these reasons, the obturator is constructed by (*a*) forming it in wax on the finished denture, (*b*) repeated corrections of its shape by frequent trials in the

mouth, and (*c*) final shaping by the addition of a layer of low-fusing wax that the muscles mold to conform to their movements. The denture is completed with a wire or cast gold retention incorporated in the denture framework and projecting from the posterior border of the finished denture where it forms the support for the obturator. This can be seen in figures 1b and 2b. Base plate wax is added to this framework in the general shape desired in the obturator. This wax is shaped by repeated trials in the mouth. The obturator must be made 10 to 12 mm. thick to provide a seal in the full range of movements made by the palatal muscles adjoining the cleft. A low-fusing paraffin or a dental impression wax can be used for this final shaping. Kerr's Iowa wax is ideal for this purpose. With the final layer of soft wax on the obturator, the patient is encouraged to talk, swallow, smoke, and engage in normal activities for a number of hours to allow the muscles to mold the wax. At the end of this time, the constrictor muscles of the pharynx should have clearly marked the posterior border of the obturator and the palatal muscles should have grooved the lateral surfaces, neatly sealing off the cleft. The denture is then reprocessed, replacing the wax with clear methyl methacrylate.

The use of clear methyl methacrylate for the obturator permits adjustments to be made in the finished denture. Muscular movements can be observed through the clear acrylic. Blanching of the tissue over the constrictor muscles of the pharynx may be observed as they impinge on the posterior border of the obturator. If the patient can tolerate it the obturator should not be shortened. If the tissues become irritated, the posterior border or the lateral borders can be carefully reduced at the points of irritation. Usually little adjustment is necessary because the obturator is constructed by a direct method. The wax pattern is molded by the soft palate and the posterior area is made concave by the molding of the superior constrictor of the pharynx. The treatment is far from completed when the finished obturator is placed in the patient's mouth. The patient with a congenital cleft palate has formed speech habits that persist after correction of the deformity. It takes much training for such a patient to make use of his new apparatus and learn to speak clearly. At this hospital where many dentures are made for the closure of clefts acquired through war wounds a dramatic comparison can be made. The patients with acquired cleft palates talk much like the patients with congenital clefts, but when they obtain obturators the correction is immediate as they were in the habit of talking correctly before their accident. Books and pamphlets are available for home-training of these patients B(*5*). In many of our general hospitals the services of a Red Cross psychologist with experience in speech training will be available.

The patient with a cleft palate presents an opportunity for the prosthodontist to render a gratifying service. In the Army a patient with this condition will be a young adult, and his age, together with the fact that an operation in the service would render his congenital defect "service connected" makes him a candidate for an obturator rather than operative repair.

### References

1. Olinger, N. A.: Prosthetic restoration of congenital cleft palate. Am. J. Orthodontics **32**: 456–468, July 1946.
2. Cantor, B. B.: Cleft palate prosthesis; treatment of case with hollowball obturator. Am. J. Orthodontics **31**: 740–743, Dec. 1945.
3. Fitz-Gibbon, J. J.: Cleft palate. Dental Digest **40**: 262–267, Aug. 1934.
4. Buchnam, E. S.: Prosthetics Department, Tufts Dental School, Boston; personal communication.
5. Blocker, T. G., Jr., and Blocker, V.: Speech training for cleft palate children. The University of Texas Press, Austin, Tex., 1948.

## Ulcer [1]

We know very little about ulcer; it would be foolhardy to deny it. The intricate interdependent relationships of the digestive glands and their secretions are not fully understood. Moreover, the agencies which influence the vulnerability of the gastric and juxtagastric mucosal surfaces to injury by acid-peptic juice, either directly or reflexly, are demanding a more intense study and clarification. That emotional urges, whether under or beyond the control of the will, may affect the susceptibility of the juxtagastric mucosal surfaces to injury by acid-peptic juice is understandable. That the behavior of emotional urges which affect unfavorably the relationship between vulnerability to acid-peptic digestion and the interaction, quantity, quality, and the time factor in the delivery—that patients who have ulcer may learn to govern these complex relationships and thus thwart the ulcer diathesis is as absurd as to believe that we can guide the movements of the stars by thinking that we can. Our objective should be to try to understand better than we do, by persistent observations, study, and inquiry, these complex interrelationships. The medical management of ulcer is sound, but its scope is limited by our very inability to accomplish consistently what we set out to do.

[1] Wangensteen, O. H.: Role of surgeon in management of peptic ulcer. Wisconsin M. J. **48**: 915–919, Oct. 1949.

# Physiological Effects of High Altitude Flying

ALONZO A. TOWNER, *Lieutenant Colonel, U. S. A. F. (MC)*[1]

IN ORDER to familiarize Air Force flying personnel with physiological stresses encountered in modern military aviation and to prepare them to meet these stresses successfully, a revitalized Air Force Physiological Training Program has been organized. This course includes the physiological effects of high altitude hypoxia, decompression sickness, and the physiology of respiration; also included are instruction in the physics of acceleration and centrifugal force to emphasize the importance of the effects of G forces and to provide instructions in ways and means of minimizing the deleterious effects of these forces through the use of G suits and proper body positioning.

Other facts emphasized are the reduction of night visual acuity when flying above 8,000 feet because the retina is susceptible to even slight degrees of anoxia; and that when flying at 12,000 feet at night, without oxygen, visual acuity drops to less than 50 percent of normal. The importance of using oxygen from the ground up to high altitude on operational night missions, as well as the proper methods for preflight dark adaptation of the eyes, is stressed.

All the possible visual sensory illusions that crew members may encounter, such as distortion of vision by plexiglass, the size-distance illusion, and autokinetic phenomena, are explained.

The purpose and use of the ejection seat is explained showing how this may provide the pilot with the only possible means of escape from an aircraft traveling at supersonic speeds or when high G forces are exerted on the pilot when the plane is out of control. The effects of conditions such as ultrasonic vibrations, windblast, and cockpit temperatures in jet aircraft are touched upon.

The greatest amount of time is spent in indoctrinating flight personnel in the proper storage, care, and use of their personal oxygen equipment and in the necessity for proper fitting of individual masks. Here emphasis is also placed on the use of walk-around and bail-out

[1] Aviation Medicine Division, Office of the Surgeon General, U. S. A. F.

equipment and in emergency procedures which may be required at great altitudes. The actual physical effects of explosive decompression are demonstrated in the low-pressure chambers and students are drilled in effective instant use of their personal oxygen equipment.

The entire physiological training program is designed to familiarize flying personnel with all the possible adverse effects to be encountered in modern military aviation and to prepare them to successfully meet these stresses and/or emergencies when they arise. The airman must be capable of recognizing emergency situations early and he must know how to make prompt and effective use of countermeasures, and personal and survival equipment. This knowledge is essential to a continued and effective program of flying safety.

The necessity for adequate aircrew training in the use of oxygen and survival equipment has been emphasized by combat commanders and flight surgeons. Now with the development of high altitude flight at supersonic speeds such training is absolutely essential. For example, the advent of pressurized cabins in aircraft is making flights to 40,000 to 50,000 feet, or higher, a commonplace occurrence. If this artificial pressurized medium were to be suddenly lost at an altitude of 60,000 feet, either by a mechanical failure or by gunfire or flak from hostile forces, the aircrew would be subject to certain death unless protected by additional items of personal clothing or equipment. These essential, lifesaving items have been developed and are fast being made available.

There are five important physical problems involved in stratospheric flights at supersonic speed.

The first and most important problem is that of hypoxia. This is closely related to a second problem, that of the escape of dissolved gases from the blood and tissues, which brings on altitude or decompression sickness (bends).

These first two conditions are related to the earth's atmosphere. The air we breathe is composed roughly of one-fifth oxygen and four-fifths nitrogen. This oxygen-nitrogen ratio in the air remains constant as we ascend toward the stratosphere but the barometric pressure decreases. At sea level the atmospheric pressure is 760 millimeters of mercury or 14.69 pounds per square inch at an outside temperature of about 60° F. At 30,000 feet barometric pressure drops to 225.6 millimeters of mercury or 4.36 pounds per square inch and the temperature drops to 48° below zero. At 60,000 feet the pressure has dropped to 54.15 millimeters of mercury or 1.05 pounds per square inch, and the temperature drops to 67° below zero.

At 60,000 feet the oxygen-nitrogen ratio of the air is unaltered but the barometric pressure is then insufficient to permit oxygenation of the blood stream with this rarefied air in the lungs. As a matter of

fact, during exposure to such rarefied air, oxygen passes from the blood stream into the alveoli and out into the air. This occurs so rapidly that the blood literally boils.

Because of the increasing rarity of the atmosphere at increasing altitudes it has been found that the average individual without supplemental oxygen will lose consciousness in 15 minutes at 20,000 feet, in 1 minute at 30,000 feet, in 20 seconds at 45,000 feet, and in 10 to 15 seconds at 50,000 feet. Forty thousand feet is considered man's oxygen ceiling, because breathing 100 percent oxygen at this altitude is equivalent to breathing ordinary air (20 percent oxygen) at 10,000 feet. Above 40,000 feet man must breathe 100 percent oxygen under pressure to compensate for the decrease in atmospheric pressure to about one-seventh of that at sea level. The Air Force requires all aircrew members to wear oxygen masks when flying above 10,000 feet for it is at and above this altitude that the deleterious effects of hypoxia become manifest. Continued and progressively severe hypoxia leads to unconsciousness and death. With cabin pressurization, and when a differential of 7.45 pounds per square inch is maintained, it is possible to fly at 40,000 feet with a cabin altitude equivalent to 9,750 feet. In this instance it is not necessary to wear an oxygen mask but one must be readily available in case of failure of pressurization. Decompression sickness (the bends, the chokes, and the creeps, depending upon which organs or tissues of the body are most severely affected) rarely occurs below an altitude of 30,000 feet. Above this altitude the "bends" can rapidly become completely disabling and will result in death if allowed to become progressively severe—but it can be completely relieved by descent to lower altitude.

Cabin or cockpit pressurization also prevents decompression sickness but a pressure suit must be worn if death is to be prevented in case of sudden loss of cabin pressurization when flying at altitudes of 60,000 feet and above. The pressure suit is, in effect, another pressurized cabin which fits closely around the pilot's body. Wearing this suit provides added assurance of safety in event of explosive or sudden decompression of the aircraft cabin.

The third problem of great importance is the extreme cold experienced at high altitudes. Exposure of body surfaces to windblast at temperatures of 50 degrees below zero will result in permanent and irreparable physical damage in a few seconds. Specially designed goggles, face masks, gloves, suits, and shoes have been developed to minimize this danger of frostbite. Some lightweight garments are completely heated by electricity.

A fourth problem is that of the expansion of body gases, under conditions of low atmospheric pressure. This will result in intestinal cramps, Barotalgia, and aerosinusitis. Gases normally found in intes-

tines will expand to seven times their volume at 40,000 feet. The pain resulting from gases trapped in defective tooth fillings or intestines becomes excruciating and in many instances has resulted in abortion of air missions through incapacitation of crew members.

Emphasis must also be placed on the importance of proper dietary habits, care of teeth, care of upper respiratory infections, and on a method of clearing the ear by carrying out a maneuver similar to Valsalva's test (closing mouth, holding nose, and blowing or swallowing).

All pilots are now given a course of physiological indoctrination during the advanced phases of their flying training.

The fifth problem is concerned with flight at supersonic speeds and is that of centrifugal force which, when applied to the body in increasing intensity, results in conditions referred to as "gray-out," "black-out," or results in unconsciousness. A man weighing 150 pounds at sea level is constantly exposed to the natural forces of gravity, referred to a force of 1 "G." During a high speed inside turn the centrifugal force may be increased to 7 G's, for example, at which time he would then be exerting a bodily pressure on his aircraft seat of seven times his weight of 150 pounds, or a total of 1,050 pounds. Similarly, the body weighs seven times its normal weight, and pooling of the blood will occur in the larger arteries and veins of the abdomen resulting in low cardiac output and cerebral anemia which leads to symptoms of gray-out, then black-out, and finally unconsciousness. When the average pilot is exposed for a period of from 15 to 20 seconds to a force of 4.2 G's he experiences gray-out, at 4.5 G's, black-out, and at 5 G's unconsciousness occurs. Frequent exposures to high G forces results in intense physical and mental fatigue. The duration of exposure to high G forces is very important; the pilot in an upright position can withstand a force as high as 10 positive G for 1 or 2 seconds.

Personnel of the Air Force Medical Service, working closely with engineers, have developed items of equipment and established procedures and techniques which protect airmen from the deleterious effects of exposure to low atmospheric pressure and low temperatures at high altitudes. The Aeromedical Laboratory at Wright-Patterson Air Force Base, Dayton, Ohio, and the Air Force School of Aviation Medicine at Randolph Air Force Base, Tex., are engaged in constant study and research on these problems.

# Laelaptid Mites as Disease Vectors

HUGH L. KEEGAN, *Captain, MSC, U. S. A.*

LAELAPTID mites are parasites of reptiles, birds, and mammals throughout the world. Recent epidemiologic studies of some of the virus encephalitides and rickettsial diseases of man indicate that detailed studies of the classification, biology, and control of parasitic mites of family *Laelaptidae* are in order. Of special interest are those species ectoparasitic on warm-blooded vertebrates, and classified, for the most part, in four subfamilies: The *Liponyssinae*, *Dermanyssidae*, *Haemogamasinae*, and *Laelaptinae*. Although nearly 400 species have been described in the 4 groups, many specie descriptions are based on examination of only single specimens, and life histories of but a few species have been accurately determined.

Various species of *Laelaptidae* normally parasitic on rodents and birds will readily attack man, their bites often producing severe dermatitis. Foremost of these, is the rat mite, *Liponyssus bacoti*, which has been reported as a pest of man in warm climates. Other parasites of rodents, which often prove troublesome, are *Liponyssus nagayoi* in Japan, and *Allodermanyssus sanguineus*, reported from several localities in the United States. *Euhaemogamasus oudemansi*, a facultative parasite, was named as the cause of an outbreak of dermatitis among American troops stationed in North Wales during the war (*1*). *Dermanyssus gallinae* and *Liponyssus bursa*, usually parasitic on domestic fowl, have frequently been reported as annoying.

Of greater importance, however, is the fact that several of the most common species of *Laelaptidae* have been shown to serve as vectors of disease. The chicken mite, *Dermanyssus gallinae*, was found naturally infected with the virus of St. Louis encephalitis by Smith, Blattner, and Heys (*2*) and with the virus of western equine encephalitis by Sulkin (*3*). Reeves and his associates (*4*) recovered the virus of western equine encephalitis from the bird mite, *Liponyssus sylviarum*, in Kern County, Calif. This mite was introduced into the United States from Europe, where it is widely distributed. Mites are of importance chiefly in transmitting the virus from bird to bird,

from which transmission to horses and to man is accomplished by mosquitoes.

Dove and Shelmire (*5*) (*6*) found *Liponyssus bacoti* experimentally capable of transmitting the rickettsial agent of murine typhus. Since that time other workers in China have reported similar results. Kodama and Kono (*7*) reported experimental infection of *Liponyssus nagayoi* with the typhus rickettsia. Ohmori (*8*) and Morisita (*9*) reported experimental infection of this mite with *Borrelia duttonii*. Yamada (*10*) reported experimental infection of the species with *Pasteurella pestis*. Rickettsialpox, most recently discovered of the rickettsial diseases, was so named because of its clinical resemblance to chickenpox. The vector was determined to be the rodent mite, *Allodermanyssus sanguineus*, by Huebner, Jellison, and Pomerantz (*11*). Phillip (*12*) found *Liponyssus bacoti* to be experimentally capable of transmitting this rickettsia, termed *Rickettsia akari* after its vector.

Great interest has been shown in *Liponyssus bacoti* in the last few years because of its role as the intermediate host of *Litomosoides carinii*, a filarial nematode parasitic in rodents. As the mites readily transmit this nematode to the cotton rat, *Sigmodon hispidus*, in the laboratory, studies of the effects of preparations used in treatment of human filariasis, caused by filariae closely related to *Litomosoides*, have been made less difficult. Other mites parasitic on lizards transmit haemogregarine protozoa similar to the malaria plasmodia affecting man, and Glaser (*13*) found organisms closely related to the rickettsiae in mites parasitic on lizards and mice. Two species currently under suspicion as vectors of murine typhus are *Laelaps nuttali*, a widely distributed parasite of rats, and *Echinolaelaps echidninus*, another rodent parasite with cosmopolitan distribution.

In view of the need for accurate information concerning the *Acarina*, it is unfortunate that there is no recent, comprehensive treatment of the group in the English language.

*Acarina* differ from other *Arachnida* in that the unsegmented abdomen is broadly united with the cephalothorax to form a saclike body, which in many species lacks all external traces of segmentation. Although the order is divided into several suborders of relatively equal status, the most common nontechnical division is that of ticks and mites. Many of the *Acarina* are free living, while others, including all known species of ticks, are parasitic during all or part of their life cycle. It is to this latter group that most attention has been given, since many of its members serve as vectors of pathogenic micro-organisms affecting man as well as domestic and game animals. Others, never directly parasitic to man, transmit agents of human disease among wild mammal and bird populations. Some species produce intense itching and dermatitis by burrowing under the skin of man,

other mammals, and birds. Dermatitis caused by bites of species not normally parasitic on man frequently occurs, and it has been reported that direct contact with some species may evoke an allergic response. Reports of intestinal, urogenital, and pulmonary parasitism of man by species of mites normally free-living are numerous in medical literature.

Ticks have been the subject of the majority of studies of the *Acarina* not only because of their large size, but primarily because of the number and severity of the diseases transmitted by them. During the war, however, when mite-borne scrub typhus and scabies became military problems, attention was directed to other members of the order.

Keys to various families and genera are scattered in the medical and zoologic literature of several languages, and many of the most important of these are available in only a few of the larger libraries in the United States. This need may be filled to a large extent by Dr. E. W. Baker of the Bureau of Entomology and Plant Quarantine, U. S. Department of Agriculture, and Dr. G. W. Wharton of Duke University, who are near completion of a comprehensive monograph in the form of a translation and a revision of an article by Vitzthum (*14*). Because of the war this publication was never distributed, and today there are less than a half dozen copies in the United States. Several workers at the University of California recently announced formation of a group to conduct a systematic study of the *Acarina*, and the number of articles on the subject in zoologic and medical journals published since the war indicates a new and active interest that should yield important results.

## References

1. Hill, M. A., and Gordon, R. M.: Outbreak of dermatitis amongst troops in North Wales caused by rodent mites. Ann. Trop. Med. **39**: 46–52, May 1945.
2. Smith, M. G.; Blattner, R. J.; and Heys, F. M.: Isolation of St. Louis encephalitis virus from chicken mites (*Dermanyssus gallinae*) in nature. Science **100**: 362–363, Oct. 20, 1944.
3. Sulkin, S. E.: Recovery of equine encephalomyelitis virus (western type) from chicken mites. Science **101**: 381–383, April 13, 1945.
4. Reeves, W. C., and others: Recovery of western equine encephalomyelitis virus from wild bird mites (*Liponyssus sylviarum*) in Kern County, Calif. Science **105**: 411–412, April 18, 1947.
5. Dove, W. E., and Shelmire, B.: Tropical rat mites, *Liponyssus bacoti* Hirst, Vectors of endemic typhus. J. A. M. A. **97**: 1506–1511, Nov. 21, 1931.
6. Dove, W. E., and Shelmire, B.: Some observations on tropical rat mites and endemic typhus, J. Parasitol **18**: 159–168, March 1932.
7. Kodama, M., and Kōno, M.: Studies on experimental transmission of virus of "eruptive fever" and "typhus" by several blood sucking insects. Kitasato Arch. Exper. Med. **10**: 99–112, April 1933.

8. OHMORI, N.: Studies on tropical rat mite; its biology and control. Taiwan Igakkai Zasshi **36**: 2608, Dec. 1937.
9. MORISITA, T.: Transmission experiments on relapsing fever with tropical rat mite, *Liponyssus* sp. Jap. J. Exper. Med. **16**: 551–558, Dec. 20, 1938.
10. YAMADA, S.: Observation on a house-infesting mite (*Liponyssus nagayoi* sp. n.) which attacks human beings, rats and other domestic animals, with brief notes regarding the possibility of plague transmission by means of this mite. Tr. Far East A. Trop. M., 8th Cong. **2**: 358, 1930.
11. HUEBNER, R. J.; JELLISON, W. L.; and POMERANTZ, C.: Rickettsialpox—newly recognized rickettsial disease; isolation of rickettsia apparently identical with causative agent of rickettsialpox from *Allodermanyssus sanguineus*, rodent mite. Pub. Health Rep. **61**: 1677–1682, Nov. 22, 1946.
12. PHILLIP, C. B., and HUGHES, L. E.: Tropical rat mite, *Liponyssus bacoti* as an experimental vector of rickettsialpox. Am. J. Trop. M. **28**: 697–705, Sept. 1948.
13. GLASER, R. W.: Intracellular "Symbionts" and "Rickettsiae." Arch. Path. **9**: 71–96, Jan. 1930, and 557–576, Feb. 1930, reprinted in Stud. Rockefeller Inst. M. Res. **73**: 465–518, 1931.
14. VITZTHUM, H. G.: Acarina, in Bronn's Klassen und Ordnugen des Tierreichs, Funfter Band, Iv Abst. 5 Buch, Lief. **1–7**: 1–1011, 1940–1943.

## Radioactive Phosphorus[1]

Radioactive phosphorus, administered intravenously, has been used clinically in order to localize and demarcate brain tumors. Its high concentration in cerebral tumors as compared with normal brain was suspected on theoretical grounds, and now appears to be adequately confirmed. A Geiger-Mueller counter of diameter comparable to that of a ventricular needle has been employed at operation in 33 cases in an attempt to localize intracranial tumors. Data adequate for localization was obtained in 29 cases, in 23 of which the tumor did not present on the surface. In one case, diffuse gliomatosis, the method was intrinsically of no value, and in two cases the method was not adequately employed. In one instance no tumor was located, and the clinical course would suggest that none was present.

[1] SELVERSTONE, B.; SWEET, W. H.; and ROBINSON, C. V.: Clinical use of radioactive phosphorus in surgery of brain tumors. Ann. Surg. **130**: 643–651, Oct. 1949.

# Insulin Atrophy of Subcutaneous Fat

## Report of a Case

KARL V. KAESS, *Commander (MC) U. S. N.*[1]

SINCE insulin was first used in the treatment of diabetes mellitus in 1922 a number of associated skin reactions have been reported. One of the most interesting of these is atrophy of the subcutaneous fat first reported by Depisch (*1*) in 1926. This phenomenon is not rare and can be found in about 7 percent of diabetics under treatment with insulin (*2*). There does not appear to be any connection between fat atrophy and the local erythematous or urticarial reactions that occasionally occur at the site of insulin injection (*3*). Fat atrophy has been reported following the use of all types of insulin (*2*) including the use of insulin in nondiabetics, for example, in the treatment of malnutrition (*4*). The amount of insulin injected is not a factor since several cases have been reported where the maximum amount injected was as little as 6 units twice daily (*5*).

### CASE REPORT

A 53-year-old white man was found to have glycosuria, about 1 year prior to admission to the hospital. A diagnosis of diabetes mellitus was subsequently made, and a diabetic diet with daily injections of 72 units of protamine-zinc insulin was prescribed. This he gave to himself into the anterolateral aspects of the thighs. On admission to the hospital, January 1949, the patient presented bilateral symmetrical depressed areas measuring about 3 by 10 inches in which the skin of the anterolateral aspects of the thighs appeared to be lying directly on the deep fascia (fig. 1). The skin in these areas was ischemic. There were also numerous striae distensae over the shoulders, chest, hips, and thighs, and a nevus araneus on the bridge of the nose. A specimen for biopsy taken from the right thigh was reported by Dr. F. D. Weidman as simple nonspecific atrophy.

On the theory that insulin fat atrophy might be caused by an Arthus phenomenon, the sites of injections were changed to the arms, alternating daily from one to the other. This explanation (Arthus phenomenon) of insulin fat atrophy was accepted, even though such tissues have never demonstrated the infiltrated and

[1] U. S. Naval Hospital, Philadelphia, Pa.

*Figure 1.—Atrophy of subcutaneous fat caused by insulin injection.*

hemorrhagic necrotic changes which one should see in such a reaction, since the specimens were taken from sites where the atrophy had already appeared. It was believed that a specimen taken early before atrophy became clinically evident might show the histologic changes of an Arthus phenomenon. Accordingly a specimen was taken from the left arm at the site of the injection after 2 months of insulin therapy, but there was no clinical evidence of fat atrophy in this region at that time. The section showed no evidence of such reaction, and was reported by Dr. Weidman as showing rather localized lymphocytic infiltration, very little edema, no eosinophilia, and no capillary hyperplasia or congestion. However, a single examination does not necessarily disprove the theory of an Arthus phenomenon since the characteristic changes might be found if serial sections of repeated specimens were examined.

This dermatosis occurs most commonly in women and children, and is seldom seen in the adult man. Insulin is not the only substance which produces fat atrophy, since it has been reported following the repeated injection of pollen (*6*) and pituitary extracts (*7*). The atrophy usually appears in about 6 months to 2 years after the injection of insulin or other solutions is begun (*3*).

According to Marble and Smith (*3*) patients with this condition have a significantly higher incidence of allergic disease (asthma, hay fever, eczema, and urticaria). Atrophy may occur at one site of injection but not at another in the same patient (*8*). It may occur at sites remote from the injection (*7*). There are no subjective symptoms acompanying the development of this change; in fact, many diabetics tend to use the same area over and over again since they find that such a site becomes analgesic (*2*).

Many theories have been advanced to explain the pathogenesis of this type of atrophy. Depisch, in his original paper, thought that it was caused by local injury to nerve fibers. Priesel and Wagner (*9*) believed that the tricresol used in preserving insulin was the cause, but Depisch gave daily injections of tricresol solution for over 2 months without producing any change. Avery (*10*) throught that repeated trauma probably damaged the delicate protoplasmic cell envelopes of the fat cells releasing the fat and eventually leading to the formation of scar tissue. Fischer (*5*) stated that a low grade inflammatory process set up by repeated injection caused a disappearance of the soft areolar tissue and replacement with scar tissue. Biopsy does

not confirm this as there are no lymphocytic foci indicative of inflammation that one sees for example in specimens taken from depressed areas in narcotic addicts (*11*). The repeated injections of narcotics produces fibrous changes in the dermis. Since both insulin and lipase come from the pancreas, lipase in the insulin was thought for a while to be the cause of the atrophy. Rabinowitch (*12*) was unable to find any lipase in concentrations of large volumes of insulin. Nicholas (*13*) believed that the strong concentration of insulin near the site of injection caused a local oxidation of carbohydrate which in turn resulted in combustion of fat. This fails to explain the occurrence of fat atrophy at remote sites.

Biopsy reports by several investigators all agree that the essential change is a simple disappearance of fat cells without any appreciable reaction. Sometimes there is a slight increase in fibrosis but the overlying epithelium is undisturbed.

## REFERENCES


1. DEPISCH, F.: Uber lokale Lipodystrophie bei lange Zeit mit Insulin behandelten Füllen von Diabetes. Klin. Wchnschr. **5**: 1965–1966, Oct. 15, 1926.
2. FERGUSON, E. A., and ALPERT, B.: Local lipoid atrophy and lipoid dystrophy; advantages of limiting sites of injections. Endocrinology **24**: 741–743, May 1939.
3. MARBLE, A., and SMITH, R. N.: Atrophy of subcutaneous fat following injections of insulin. Proc. Am. Diabetes A. **2**: 171–186, 1942.
4. BLOTNER, H.: Fatty atrophy following insulin injection in nondiabetic malnutrition. Endocrinology **23**: 233–236, Aug. 1938.
5. FISCHER, A. E.: Frequency of atrophy of subcutaneous fat following injection of insulin. Am. J. Dis. Child. **38**: 715–720, Oct. 1929.
6. FRANCIS, N.: Localized atrophy of subcutaneous fat after repeated injections of grass pollen. Ann. Allergy **2**: 344–345, July–Aug. 1944.
7. FREI, W.: Insulin atrophy of subcutaneous tissue. Arch. Dermat. & Syph. **37**: 534–535, March 1938.
8. SPIEGELMAN, A. R.: Selective localization of insulin atrophy. New York State J. Med. **44**: 2723, Dec. 15, 1944.
9. PRIESEL, R., and WAGNER, R.: Uber lokale Lipodystrophie nach Insulin injectionen. Ztschar. f. Kinderh. **46**: 453–456, 1928.
10. AVERY, H.: Insulin fat atrophy a traumatic atrophic panniculitis. Brit. M. J. **1**: 597–599, March 30, 1929.
11. MENTZER, S. H., and DUBRAY, E. S.: Fatty atrophy from injections of insulin California & West. Med. **26**: 212, Feb. 1927.
ing injections of insulin. Canad. M. A. J. **21**: 67–68, July 1929.
12. RABINOWITCH, I. M.: Case of extensive atrophy of subcutaneous fat follow-
13. NICHOLS, J. B.: Atrophy of subcutaneous fat associated with insulin injections. Am. J. M. Sc. **180**: 90–95, July 1930.

# Morphine Withdrawal

## Report of a Case

CHARLES T. BROWN, *Major, MC, U. S. A.*[1]

ALTHOUGH morphine addiction as a primary cause for admission to an Army hospital is rare, a patient is occasionally seen who has both a psychologic and physiologic dependency on morphine or some other narcotic because of a painful, chronic disease. In such an instance, the time usually comes when the patient is free of pain, and except for the ordeal of the abstinence syndrome would be eager to free himself of addiction.

The case report that follows illustrates a method by which a patient may be rapidly relieved of addiction to morphine with a minimum of discomfort through the use of Methadon (6-dimethyl-amino-4, 4-diphenly-3-heptanone hydrochloride). This drug was developed by the Department of Commerce, and intensive research at the Public Health Service Hospital at Lexington, Ky., has demonstrated its effectiveness in the treatment of morphine addiction. There, it has been demonstrated that Methadon is specific for the morphine abstinence syndrome. That the drug might be useful in the withdrawal of other narcotics having a morphinelike action is a rational conclusion. Although Methadon also has definite addiction liability, the abstinence syndrome induced by its continued use is of less intensity than that produced by the withdrawal of morphine. Prior to the introduction of Methadon, almost every known drug was used in an attempt to carry the addict through the agonizing misery of the abstinence syndrome. In this new drug, we have the most effective substitutive therapeutic agent yet employed.

### CASE REPORT

A 60-year-old man who had been taking morphine continuously for 41 years was admitted to the neuropsychiatric service. He had initially been admitted to the tuberculosis service, where it was determined that he had advanced pulmonary tuberculosis with extensive cavitation. On being informed of the nature of his disease, he expressed a desire to be relieved of his addiction to

[1] Neuropsychiatric Service, Fitzsimons General Hospital, Denver, Colo.

morphine. He undoubtedly felt, and rightly so, that his chances of prolonged survival might be enhanced if he were relieved of this addiction.

His personality during his early life might be described as emotionally unstable and hedonistic. In 1908, following an alcoholic debauch, he was injudiciously and repeatedly given morphine by a physician. Pleased by the soothing effects of the drug, he learned its identity and from that time on administered it to himself. Over a period of years, his daily ration of morphine often amounted to 4 gm. This enormous dosage was curtailed by the enactment of the Harrison Narcotic Act in 1915, and in the years that followed, his daily maintenance dose was of necessity reduced. He tried, in 1923, to relieve himself of addiction in a sanitarium, where judging from his description, hyoscine was used as a substitutive agent. He was unable to withstand the rigors of the treatment employed, left the institution, and resumed the use of morphine. From that time on his daily dose of morphine was about 0.8 gm. In the past few years because of difficulty in obtaining the drug, he took 0.25 gm. daily subcutaneously in 4 doses. This was the dose to which he was accustomed at the time of his admission to this hospital. On transfer to the neuropsychiatric service, he was given this dose of morphine for the first 2 days in order that he could become used to his new surroundings, and that rapport with his physician might be obtained before starting withdrawal.

Then Methadon 20 mg. along with 16 mg. of morphine sulphate was given every 4 hours for 3 days. The Methadon and morphine were given in separate syringes. On the sixth day, morphine was discontinued, and the patient was given 20 mg. of Methadon every 4 hours. This was continued until the ninth day, when the dose of Methadon was reduced to 15 mg. every 4 hours. For the next 3 days the dose was reduced each day until on the twelfth day, he was receiving only 5 mg. of Methadon at each injection. The drug was then discontinued and for 3 succeeding days, he received injections of sterile water at his accustomed hour of medication. In the last 2 days of his withdrawal period, the cumulative effects of the Methadon gradually subsided, and the patient became restless. At no time, however, did he complain of any discomfort, and he proved most cooperative throughout the period of treatment. For insomnia, of which he complained for a few nights after the discontinuance of the Methadon, he was given 1.3 gm. of chloral hydrate at bedtime. This was repeated during the night if necessary.

On the twenty-first day following the beginning of withdrawal, he was transfererd back to the tuberculosis service for further observation. He was followed daily after his transfer, and aside from minor complaints of restlessness and general weakness, he exhibited no discomfort. He at no time asked for any type of medication, and stated that he no longer felt the desire for morphine. He was discharged after 2 months of hospitalization at his own request in order that he might return home to settle some business affairs. Three months following his discharge from this hospital, he stated in a personal communication that he felt fairly well and had no desire to resume the use of narcotics.

## DISCUSSION

The most important factor in the withdrawal of a narcotic from an addict is the physician-patient relationship. No physician, other than the one undertaking the withdrawal, should participate in the treatment except in an emergency. The nurses and attendants must be especially instructed in regard to the treatment. Most past failures

859434°—50——6

in the treatment of morphinism have been caused by a lack of understanding and knowledge in the handling of this type of patient. Drug addiction may be as strange and just as difficult to manage as schizophrenia. The abstinence symptoms are both psychologic and physiologic in nature. The patient is unfortunate rather than depraved and, therefore, worthy of kindness and consideration. A complete physical examination, along with comprehensive psychologic studies, is important. Every effort should be made to exclude the possibility of painful, chronic organic disease. There is no need in every case to try to relieve the patient of an addiction if there exists a sound physical basis for it such as the terminal stage of cancer.

The physician should tell the patient that he intends to relieve the drug dependency with as little discomfort as possible. The patient should be assured that he is in friendly and understanding hands, and that nothing will be required of him that he cannot endure. On the other hand, he should not be promised absolute freedom from discomfort any more than an obstetrical patient should. After a day or two of orientation on the ward the period of detoxification may be started. The symptoms of withdrawal usually become manifest from 4 to 8 hours after the last accustomed dose of the drug. By the twelfth hour they become almost unbearable, and from the twenty-fourth to the forty-eighth hour their intensity increases. From the forty-eighth to the seventy-second hour is the period of greatest agony, if the drug is completely withheld. From this period on, the intensity of the pains of withdrawal gradually subside, and although the patient may complain of various symptoms, they are as a rule bearable. The intensity of the withdrawal symptoms does not depend on how much drug the patient has been taking nor on how long he has been taking it. Advancing age is no contra-indication to treatment as was formerly believed.

Addiction to narcotics is indicative of a basic personality disorder. There are both psychologic and physiologic aspects to addiction. Oscar Jennings, a physician and a morphine addict, contributed more than anyone else to the problem in the latter part of the nineteenth century. Addiction to opium, and its derivatives, had at that time become rather widespread in the United States, having first made its appearance about 1860. Jennings was the first to postulate that there was a strong psychologic component to addiction in addition to the physiologic and toxic symptoms. George Pettey, in 1912, confirmed the earlier observations of Jennings. This was followed by the sound observations and conclusions of Alexander Lambert in 1915. Lambert stated that the withdrawal symptoms in morphine addiction represented "a quasi-hysterical response to a balked imperative desire." He also recognized certain reversible physiologic alterations in the

addict and stressed the importance of physical rehabilitation along with concerted efforts toward the improvement of the impoverished nutrition usually accompanying narcotic addiction. The patient throughout treatment should be given intravenous fluids when indicated and a high calorie diet and vitamins. The violent purging that was considered an important part of treatment in earlier years is to be deplored. Recent studies have proved the value of modified insulin therapy in restoring autonomic nervous imbalance on the completion of drug withdrawal. The use of insulin, however, does little to relieve withdrawal symptoms in the acute phase. The follow-up with intensive psychotherapy, prolonged supervision, and guidance appropriate to the needs of the patient is the most important part of the treatment. If the patient is expected to remain free from narcotic addiction following a successful withdrawal, this is the most important aspect of the problem.

## Blood Loss During Surgery[1]

Blood lost during thoracic operations can be measured by the increase in weight of the gauze sponges used. This method gives a continuous record of blood loss during the course of an operation and so permits simultaneous accurate replacement by blood transfusion.

There is no correlation between the amount of blood lost and the simultaneous changes in hematocrit, hemoglobin, and plasma protein before and after operations. Even loss of 500 cc. of blood is of vital importance to the injured or sick person.

There is no clinical sign or test of impending shock and clinical evidence of shock does not appear until approximately 1,200 cc. of blood are lost. During operative procedures replacement of blood volume must not be delayed until shock becomes evident.

---

[1] MILLER, B. J.; GIBBON, J. H., JR.; and ALLBRITTEN, F. F., JR.; Blood volume and extracellular fluid changes during thoracic operations. J. Thoracic Surg. 18: 605–615, Oct. 1949.

# Thiomerin

## A Mercurial Diuretic for Subcutaneous Administration

DALLAS E. BILLMAN, *Lieutenant, junior grade (MC) U. S. N.*[1]
HENRY R. COOPER, *Lieutenant (MC) U. S. N.*[1]
RALPH C. PARKER, JR., *Commander (MC) U. S. N.*[1]

IN THE last 2 years investigations using BAL and related compounds have been reported and have led to modifications of mercurial diuretics. A recently developed compound, Thiomerin (MT6), a mercaptide complex, was studied clinically to test its diuretic effectiveness and to evaluate the claim that it can be safely administered by subcutaneous injection.

### METHOD

Patients in congestive heart failure were picked at random for the study. Thiomerin in doses of 1 or 2 cc. was administered subcutaneously; in addition, a low sodium diet, ammonium chloride, and an unrestricted fluid intake was prescribed. If taking digitalis on admission, a maintenance dosage was continued, but no digitalization was begun while the patients were receiving Thiomerin. Weight change on this regime was used as objective evidence of the effectiveness of the treatment.

### RESULTS

Forty-one subcutaneous injections of 1 cc. each were given: the average weight loss within 24 to 48 hours was 3.5 pounds per injection. Forty-nine 2 cc. subcutaneous injections resulted in an average weight loss of 4.4 pounds within 24 to 48 hours following each injection. No weight change was noted with five injections and there was an average weight gain of 0.33 pound per injection in seven instances. No general toxicity was noted in this series. Three injections resulted in minor local reactions, consisting of tenderness and slight erythema, together with minimal induration in one case. All evidence of local irritation disappeared within 24 to 48 hours.

[1] U. S. Naval Hospital, Bethesda, Md.

It was the opinion that diuresis obtained with Thiomerin was approximately the same as that resulting from other mercurial diuretics used under similar conditions on the same ward.

## DISCUSSION

Since the discovery, in 1920, of the diuretic action of certain organic mercurial compounds, research has been directed toward enhancing this diuretic effect while decreasing the undesirable side reactions. A mercury-urea compound, merbaphen, was introduced in 1920 (*1*), Salyrgan (a mercury-salicylate) in 1924 (*2*), and Mercupurin (a mercury-theophylline compound) in 1928 (*3*). These preparations are available in the form of ampules, tablets, and suppositories. The intravenous route of administration has long been recommended as the preferable method because of the risk of local irritation when used intramuscularly. Frequent and severe local reactions have prohibited general use of the subcutaneous route of administration. However, due to an occasional case of sudden death following intravenous use of the mercurials, the intramuscular route has been gaining favor. Mercupurin is probably the drug of choice because it produces fewer undesirable local effects.

In 1947 Lehman (*4*) demonstrated that the general toxicity, as reflected in electrocardiographic changes of mercuric chloride, Salyrgan-theophylline, Mercuzanthin, Mercuhydrin, and MT6 (the disodium salt of N (*γ*-carboxymethylmercaptomercuri-B-methoxy) propyl camphoramic acid) decreased in the order named. The latter preparation produced no electrocardiographic changes in doses 160 times the maximum tolerated dose of Mercuhydrin and 400 times the maximum tolerated dose of mercuric chloride. The decrease in the toxicity of this new mercurial compound is thought to be due to the relatively greater stability of this mercaptide as compared with the theophylline complex (*4*). The development of a mercurial diuretic in the mercaptide form occurred in the following manner:

Experimental work has shown that some mercurial compounds and trivalent arsenicals inactivate certain enzymes which contain sulfhydryl groups (*8*). The combination of the mercurials and the sulfhydryl radicals apparently forms a stable mercaptide which prevents the action of the enzyme. It is believed that mercurial diuretics inactivate specific enzymes of the renal tubules and thus prevent the reabsorption of sodium, thereby bringing about a sodium and water diuresis (*5*). Reaser and Burch (*6*) in studies utilizing radioactive $Na^{22}$, have shown that a sodium diuresis precedes the water diuresis by 2 to 4 hours after the administration of a mercurial diuretic.

The discovery that the di-thiol compound, BAL, prevented the diuresis caused by mercurial diuretics opened a new line of investiga-

tion (*8*). BAL supposedly forms a stable mercaptide with mercurials and thus prevents the toxic effects of mercury ions, or the inactivation of certain enzymes as indicated above. It has been demonstrated that the mono-thiol compounds, thioglycollic acid and glutathione, will also inhibit the diuretic effect of mercurials but in much larger doses than the di-thiol compound BAL (*8*).

It is recognized that sudden deaths resulting from the intravenous injection of mercury compounds are due to their cardiotoxic effects. By using the electrocardiograph, Lehman (*4*) measured the cardiotoxic effect of several mercury compounds as listed above. The most significant electrocardiographic changes consisted of abnormalities of conduction, widening of the QRS complex, was a consistent measure of toxicity. It was adequately demonstrated that the mercaptide MT6 formed by combining an organic mercurial, N(y-hydroxymercuri-B-methoxy) propyl camphoramic acid, with an aqueous solution of sodium thioglycollate, was distinctly less toxic than mercuric chloride or the mercury-xanthine compounds.

A further step toward a better mercurial diuretic was the demonstration by Farah and Maresh (*5*) that in contrast with di-thiol BAL, the monothiols, cysteine, and glutathione, in doses fully active against the cardiotoxic effects of Mersalyl, did not decrease the diuretic effect of this mercurial. Thus, the development of the theoretically ideal mercurial diuretic appeared to be closely approached by the combination of an organic mercurial with one of the mono-thiols, thereby preventing the cardiotoxic effects while preserving the diuretic action.

It has been previously shown that the combination of a mercurial and theophylline decreased the local irritative effects of the diuretic (*11*)—however, not to the extent that it could be generally administered by the subcutaneous route. As the mercaptide form is much more stable than the theophylline complex, it is to be expected that the new mercurial compound MT6 would have less local toxicity than those mercury-xanthine compounds now in general use. That this is true is attested by the work of Borg and Craig (*9*) and the present brief clinical series. The mercaptide, Thiomerin (MT6), is an effective mercurial diuretic and can be used in adequate clinical doses by subcutaneous injection. The desirability of this manner of using a mercurial diuretic is obvious; for example, a patient who is on a maintenance biweekly or weekly dose may use it himself in the fashion that the diabetic uses his insulin. The injection, of course, should not be given in an endematous area.

## SUMMARY

A short clinical series is presented showing the effect of the new mercaptide diuretic Thiomerin (MT6) given by subcutaneous in-

jection, and a discussion of the mode of derivation of this compound is included.

Thiomerin is an effective mercurial diuretic with minimal general and local toxicity. It can be administered with safety by the subcutaneous route in ordinary clinical doses and is considered safe to dispense for self-administration.

## References


1. Saxl, P., and Heilig, R.: Diuretic effect of "novasurol" and other mercury preparations. Wien. klin. Wchnschr. **33**: 943, Oct. 21, 1920.
2. Brunn, F.: Salyrgan, a new diuretic injection. Wien. klin. Wchnschr. **37**: 901–902, Sept. 11, 1924.
3. von Issekutz, B., and von Vegh, F.: Uber die diuretische, Wirkung organischer Quecksilberverbindungen. Arch. f. exper. Path. u. Pharmakol. **138**: 245–255, 1928.
4. Lehman, R. A.: Further studies on acute toxicity of mercurial diuretics. Proc. Soc. Exper. Biol. & Med. **64**: 428–433, April 1947.
5. Farah, A., and Maresh, G.: Influence of sulfyhydryl compounds on diuresis and renal and cardiac circulatory changes caused by mersalyl. J. Pharmicol. & Exper. Therap. **92**: 73–82, Jan. 1948.
6. Reaser, P. B., and Burch, F. E.: Radiosodium tracer studies in congestive heart failure. Proc. Soc. Exper. Biol. & Med. **63**: 543–546, Dec. 1946.
7. Earle, D. P., Jr., and Berliner, R. W.: Effect of 2, 3-dimercaptopropanol on diuresis. Am. J. Physiol. **151**: 215–220, Nov. 1947.
8. Handley, C. A., and Laforge, M.: Effect of thiols on mercurial diuresis. Proc. Soc. Exper. Biol. & Med. **65**: 74–75, May 1947.
9. Borg, J. F., and Craig, D. M.: Experiences with a mercurial diuretic for subcutaneous injection. J. Lab. Clin. M. **33**: 1635, 1948.
10. DeGraff, A. C.; Nadler, J. E.; and Batterman, R. C.: Study of diuretic effect of mercupurin in man. Am. J. M. Sc. **191**: 526–538, April 1936.
11. DeGraff, A. C., and Lehman, R. A.: Acute toxicity of mercurial diuretics. J. A. M. A. **119**: 998–1001, July 25, 1942.

# Accessory Membranous Naso-lacrimal Duct

## Report of a Case

ADRIAN J. DELANEY, *Captain (MC) U. S. N.*[1]
SAMUEL H. OLIVER, *Commander (MC) U. S. N.*[1]

CONGENITAL abnormalities are often first noted incidental to some other pathologic condition. Most tests on ophthalmology give only a very brief description of the developmental and anatomical relationships of the lacrimal passages. In order to evaluate the reasons for a congenital or developmental anomaly, a review of the embryology, development, and anatomical relations of a system is necessary.

Onodi (*1*), in his remarkable volume, describes the anatomical relations of the nasolacrimal canal and duct as follows:

The lachrymal fossa is directly continuous with the osseous naso-lachrymal canal which is formed by three bones: namely, the superior maxilla, the lachrymal, and the inferior turbinal bones. The largest share in its formation is taken by the superior maxilla and is supplied by the posterior border of the frontal process and by part of the body of that bone. The gutter seen in this situation is converted into a canal by the lachrymal bone above and by the lachrymal process of the inferior turbinal below. The canal so formed has its lower opening, the nasal orifice or ostium lachrymale, immediately below the line of attachment of the inferior turbinal bone. The length of the osseous naso-lachrymal canal usually does not exceed 10 or 12 mm. * * *but may vary from 10 to 24 mm. * * *. In diameter it measures 3 to 4 mm. In its topographical relations the canal corresponds to the whole vertical extent of the middle meatus, medial to the maxillary antrum * * *.

Onodi (*1*) remarks that:

As seen in the dry skull the naso-lachrymal canals diverge as they pass down * * * . Looked at in profile they will be seen to pass downwards and rather backwards from the angle of the orbit towards the interval between the second bicuspid and the first molar teeth * * * . As it runs downwards and outwards its lateral deviation corresponds, according to Arlt, to a line drawn from the point where the medial (internal) palpebral ligament crosses the sac, to the naso-labial fold.

---

[1] U. S. Naval Hospital, Bethesda, Md.

Three positions for the inferior meatal opening of the nasolacrimal duct are described by Spaeth (*2*);

the first, in the roof of the inferior meatus; a second, in the side walls of the inferior meatus; and a third, as a small canal in front of the inferior meatus. The first two positions are the usual, the third rather rare.

The nasolacrimal duct arises in 12 mm. embryos (*3*) as a ridgelike thickening of the epithelial lining of the nasolacrimal groove. This thickening becomes cut off, and as a solid cord sinks into the underlying mesoderm. There is disagreement among many of the earlier writers as to the development of the efferent lacrimal ducts, particularly the canaliculi.

Fischer (*4*) in an extended review of this subject and from his own observations, believes that the development of the lacrimal duct begins in the 11 mm. embryo as a triangular thickening formed on the superficial ectoderm of the nasolacrimal groove. From this epithelial thickening there soon develops a peculiar processlike invasion in the embryonic connective tissue which is the anlage of the system. He states that it can be shown that the anlage of the lacrimal duct is developed, neither by an outgrowth of solid epithelial plate from the fundus of the nasolacrimal groove (Born), nor by bridging of the nasolacrimal groove by cells of the superficial ectoderm (Erdl-Coste). He further states that the end of the anlage, near the eye from the very beginning, shows changes which lead to the development of the lacrimal canaliculi and there is no proof for the opinion of Speciale-Cirincione (*5*) that the lacrimal ducts are formed by two epithelial strands, originally developed from an epithelial plate of the nasolacrimal groove and later partly fused secondarily.

At any rate all the efferent lacrimal duct system is at first blocked out in solid epithelium (*6*) and canalize just before birth.

According to Mann (*6*) it is "therefore obvious that defects of three types are to be expected. Either the passages will not develop, or the epithelial buds from them will pursue an abnormal course, or the apparatus will fail to canalize."

The following anomalies of the efferent lacrimal system have been described: (*a*) membranous occlusion of the punctum (*4*); (*b*) absence of punctum and canaliculi (*1*) (*4*); (*c*) occlusion of the punctum and canaliculi (*4*); (*d*) duplication of canaliculi (*1*) (*4*) (*7*); (*e*) triple canaliculus and punctum (*1*) (*4*) (*7*); (*f*) quadruple lower punctum (*4*); (*g*) formation of groove near the canaliculus (*4*) or open cleft in place of the lower canaliculus (*8*); (*h*) elongation of the lower canaliculus as in hypertelorism (*4*); (*i*) absence of the lacrimal sac (*4*); (*j*) duplication of the lumen of the lacrimal sac (*1*) (*4*); (*k*) duplication of the lacrimal sac (*4*); (*l*) diverticulum of the lacrimal sac (*4*); (*m*) cavernation instead of the lacrimal sac (*4*) (*11*); (*n*)

congenital fiistula of the lacrimal sac (*4*) (*6*) (*10*) (*12*); (*o*) membranous occlusion of the inferior meatal opening of the nasolacrimal duct (*1*); (*p*) failure of canalization of the nasolacrimal duct (*1*); (*q*) folds and valves in the nasolacrimal duct (*1*); and (*r*) excretory duct of the lacrimal gland emptying on the cheek (*9*).

Fistulas of the lacrimal sac, according to most writers, are not uncommon. In most of these cases there is a tiny cleftlike opening on the skin located nasally and below the inner canthus, that is, below the medial canthal ligament. The fistulas may or may not be bilateral and symmetrical. The opening may lie alongside the caruncle (*4*). The fistulas may open into the sac or they may be blind.

Authors vary in their opinions concerning the origin of the fistulas. Fischer (*4*) states that Elschnig believed these to be due to a separation of the epithelial anlage of the lacrimal duct from the superficial ectoderm; that Lohlein believed them to be supernumerary misplaced canaliculi. Fischer (*4*) maintains that since in 16 mm. embryos, the anlage of both canaliculi is present, and is in contact with the superficial ectoderm by means of an epithelial pedicle emanating from the center of the anlage, then the fistula must be simply a relic of the early epithelial invagination of the lacrimal groove especially since the opening on the skin is, for the most part, in the predetermined location below the internal canthus.

## CASE REPORT

R. R. Z., a 19-year-old white man, was admitted for treatment of an injury in the right maxillary region which occurred while he was playing football. The soft tissue overlying the right maxilla below the inferior orbital rim was red, indurated, and tender and was treated as traumatic cellulitis. During repeated examinations, a small cleft opening on the skin 3 mm. below the inferior canaliculus on the right was observed from which a small

*Figure 1.*

*Figure 2.*

amount of clear fluid could be expressed. The patient had not been aware of any discharge, however, his mother had noted this since birth. No one in the family had had a similar anomaly. Since this structure could have been a fistula from a deep-seated cystic structure and which could account for the prolonged inflammation, investigation of its nature was indicated.

Roentgenograms of the sinuses were essentially negative. A probe was passed into the normal nasolacrimal system (right) and lipiodol solution injected into the fistula opening. There are two entirely separate nasolacrimal membranous systems in the bony canal which open into the inferior meatus (figs. 1 and 2).

## COMMENT

The evidence indicates the presence of an entirely normal nasolacrimal system on the right and an accessory, nonfunctional, though patent, second system in the form of a fistula 3 mm. below the inferior canaliculus on the same side. This extremely rare congenital anomaly most likely developed as a result of a simple duplication of the epithelial thickening on the superficial ectoderm of the nasolacrimal groove and its development is closely analogous to that of fistulas of the lacrimal sac because of its predetermined ostium location below the internal canthus.

## REFERENCES


1. ONODI, A.: The Relations of the Lachrymal Organs to the Nose and Nasal Accessory Sinuses. William Wood & Co., Baltimore, Md., 1912, pp. 46 and 53.
2. SPAETH, E. B.: Principles and Practice of Ophthalmic Surgery. 3d edition. Lea and Febiger, Philadelphia, Pa., 1944 p. 109.
3. AREY, L. B.: Developmental Anatomy. W. B. Saunders Co., Philadelphia, Pa., 1925. p. 305.
4. FISCHER, F.: Die Fehlbildungen der Ableitenden Thränenwege und ihre formale Genese. Zeitschrift f. Augenheilkunde 94: 1–11; 95: 152–161, 1938.
5. SPECIALE-CIRINCIONE, F.: Lo sviluppo delle vie lacrimali nell'uomo. Ann. di. ottal. e. clin. ocul. 57: 435–493, June–July 1929.
6. MANN, I.: Developmental Abnormalities of the Eye. Cambridge University Press, London, 1937, pp. 414–416.
7. CARVILL, M.: Congenital fistulae of lachrymal canaliculi. Arch. Ophth. 38: 585, 1909.
8. COSMETTATOS, G. F.: Ueber einige angeborene Anomalie der Thränenwege. Arch. f. Augenheilkunde 55: 362–371, 1906.
9. VON BAMBERGER: Beitrag zu den Congenitalen Anomalien der Thränenwege. Deutsches Arch. f. Klin. Med. 69: 618–621, 1900–01.
10. DALEN, A.: Ein Fall von doppelseitiger, kongenitaler Tranensackfistel. Mitt. a. d. Augenklin. d. Carolin. Med. Chir. Inst. Zu Stockholm, 5: 1–7, Jena 1904.
11. WERNCKE, T.: Ueber eine bisher nicht beobachtete Bildungshemming des Tränensackes. Klin. Monatsbl. f. Augenh. 47: 83, 1909.
12. TORTI, M.: Contributo allo studio fistole congenite del sacco lacrimale. Rassegna ital. d'ottal. 15: 69–76, Jan.–Feb. 1946.

# Planning, Organization, and Administration of a Large Dental Prosthetic Activity During Wartime

ALVIN H. GRUNEWALD, *Commander (DC) U. S. N.*

THIS report outlines the work load, personnel, facilities, treatment plan, and procedures necessary for setting up and operating a large dental prosthetic activity during wartime and also reflects the policy of providing the greatest good for the greatest number rather than the highest degree of excellence for a chosen few.

This report is based principally upon experience gained in the planning, construction, organization, and administration of the prosthetic department at one of the Navy's largest training centers (Great Lakes, Ill.) during World War II. It is felt that the basic information developed through that experience should be of value to anyone having a similar assignment in the future and therefore should be made available to others. Examples of survey findings and of procedures developed at that activity are included.

## WORK LOAD

The first thing to be determined in planning a prosthetic activity is the work load. This can be found by determining the number of recruits to be inducted at the activity and the number and types of prosthetic restorations that will be required.

*Prosthetic requirements of recruits.*—The shortage of facilities at times necessitated limiting prosthetic treatment to those recruits who had less than the equivalent of three bicuspids in occlusion. Using this as a basis, it was found that the following percentages of all recruits inducted required treatment during a 12-month period in 1943 and 1944.

| Month: | *Percent* | Month: | *Percent* |
|---|---|---|---|
| August | 4.9 | February | 7.5 |
| September | 6.9 | March | 9.9 |
| October | 6.2 | April | 7.5 |
| November | 6.9 | May | 6.9 |
| December | 8.5 | June | 5.2 |
| January | 8.8 | July | 3.4 |

Average monthly percentage for the year was 6.9.

These figures would have to be increased approximately 10 percent (e. g., for August—increased from 4.9 percent to 14.9 percent) if all recruits requiring some type of prosthetic treatment are included.

The fluctuation in percentage from month to month was due to the difference in the number of men of different age groups inducted and the areas from which they came during a given month. It appeared that the prosthodontia requirements were highest for men inducted in the eastern part of the United States, with men from the South running a close second.

The percentage of 4,000 recruits requiring prosthodontia, by age groups, was as follows:

| Age: | *Percent* | Age: | *Percent* | Age: | *Percent* |
|---|---|---|---|---|---|
| 17 | 0.05 | 27 | 4.00 | 37 | 5.48 |
| 18 | 4.38 | 28 | 4.13 | 38 | 3.88 |
| 19 | 16.43 | 29 | 4.55 | 39 | .90 |
| 20 | 3.95 | 30 | 5.80 | 40 | .04 |
| 21 | 1.90 | 31 | 5.48 | 41 | .05 |
| 22 | 1.90 | 32 | 4.98 | 42 | .08 |
| 23 | 2.08 | 33 | 5.65 | 43 | .05 |
| 24 | 2.10 | 34 | 5.15 | 44 | .05 |
| 25 | 3.18 | 35 | 4.95 | | |
| 26 | 3.93 | 36 | 4.98 | | |

The average number of dentures required was 1.6 per man.

The distribution of the various types of treatment required was:

| Types: | *Percent* | Types: | Percent |
|---|---|---|---|
| Full upper dentures | 31.3 | Partial lower dentures | 31.8 |
| Full lower dentures | 11.5 | Miscellaneous | 1.4 |
| Partial upper dentures | 24.0 | | |

Most patients required a combination of upper and lower dentures. The distribution of dentures, by patients, was as follows:

| Denture combinations: | *Percent of Patients* |
|---|---|
| Single full upper cases | 11.6 |
| Single full lower cases | .5 |
| Full upper and full lower dentures | 18.0 |
| Full upper and partial lower dentures | 21.5 |
| Full lower and partial upper dentures | .3 |
| Single partial upper dentures | 17.8 |
| Single partial lower dentures | 9.3 |
| Partial upper and partial lower dentures | 21.0 |

*Prosthetic requirements of station personnel.*—The prosthetic requirements of personnel assigned to the station for duty can be disregarded in determining the facilities needed. These are nominal compared to those of the recruits, and can easily be met during slack periods in induction.

*Preprosthetic requirements—Oral surgery.*—Recruits in need of prosthetic treatment required an average of 9 extractions per man. Conservative surgical preparation of the mouth was performed routinely; in addition to this preparation and extractions the amount of minor oral surgery was nominal.

*Preprosthetic requirements—Operative.*—It was found that of those recruits requiring some form of partial prosthesis, an average of 7 out of 10 recruits (70.8 percent) required operative treatment. These latter patients required an average of 10 restorations per man, mostly two-surface or larger.

The oral surgical and operative requirements also varied with the area from which recruits were inducted, and were the highest in recruits from the eastern section of the country.

## PERSONNEL

After making an estimate of the work load, the next thing to be considered is the number of dental officers and corpsmen needed in the entire prosthetic set-up, including the oral surgery and operative clinics assigned to do the necessary preprosthetic treatment.

The personnel requirements of the prosthetic department proper should be divided into those of the (*a*) clinic, (*b*) laboratory, and (*c*) administrative sections.

(*a*) *Clinic personnel required:* The number of dental officers required in the clinic section depends upon the prosthetic technics to be employed and the capabilities of the dental officers. A dental officer previously engaged in the general practice of dentistry, and possessing a fair degree of knowledge and experience in prosthetics, can complete from six to eight dentures per day, provided that not over half of these are full dentures and that all laboratory procedures are completed for him in a satisfactory manner within a specified time.

It is further essential that (*a*) the simplest technics commensurate with good dentistry be employed, (*b*) the dental officer be permitted to work according to a definite, established schedule, and (*c*) the activity be so organized that none of his time need be devoted to administrative details. One Dental Corps man should be assigned to assist each dental officer in the clinic section.

(*b*) *Laboratory personnel required:* The personnel required in each section for a prosthetic laboratory designed to complete work for a company of 120 men (up to 200 dentures) per day was as follows (the average technician working under Service wartime conditions is approximately one-half as efficient as the average technician working in a civilian laboratory):

| At sit-down benches (by procedure): | Number of men |
|---|---|
| Baseplates, occlusal rims, and wax for trial set-ups | 9 |
| Bent wire skeletons | 18 |
| Gold castings (and bent wire skeleton when required) | 3 |
| Set-up for full and partial dentures | 18 |
| Relief outline and postdam | 1 |
| Tinfoil | 6 |
| Special acrylic resin work | 3 |
| Wax and rough finish | 24 |
| Repairs | 2 |
| | 84 |

| At stand-up benches (by procedure): | |
|---|---|
| Plaster bench (fix alginate impressions; pour; separate and trim models; mount on articulator) | 11 |
| Invest, boil out sticky wax, and preheat partial skeletons for soldering | 2 |
| Occlusal matrix and flasking | 6 |
| Wax boil-out | 2 |
| Acrylic packing | 4 |
| Acrylic curing | 1 |
| Deflasking | 4 |
| Polishing | 8 |
| Flask, tray, and articulator clean-up | 2 |
| | 40 |

| Laboratory supervision and administration (by duty) | Number (of men) |
|---|---|
| Case recorders | 2 |
| Laboratory stockroom | 1 |
| Tooth and gold issue | 1 |
| Chief supervisor | 1 |
| Bent-wire supervisor | 1 |
| Setup supervisor | 1 |
| Wax, rough-finish, and polish supervisor | 1 |
| | 8 |
| Total technicians | 132 |
| Dental officers in laboratory: | |
| Officer in charge | 1 |
| Dental officer—assistant | 1 |
| Total officers | 2 |

The number of men assigned to each section of a prosthetic laboratory will vary with the ability of the technicians employed, the clinic and laboratory technics adopted, and the percentage of full dentures

to partial dentures. Generally four or five prosthetic technicians are required for each dental officer at a dental chair.

(*c*) *Administrative personnel required* for a prosthetic activity designed to treat 120 men per day, was as follows:

| Enlisted personnel (by duty) | Number (of men) |
|---|---|
| Case runners | 3 |
| Form L and file clerks | 2 |
| Typists | 3 |
| Case numbering | 1 |
| Telephone operator | 1 |
| Information and appointments | 2 |
| Property and personnel | 1 |
| Maintenance and repair | 1 |
| | 14 |

| Dental officers (by duty): | Number (of men) |
|---|---|
| Officer in charge | 1 |
| Clinic supervisor | 1 |
| Examinations and treatment coordination | 2 |
| Explorative operative procedures and X-rays | 1 |
| Property and personnel | 1 |
| Dental officers providing prosthodontia | 28 |
| | 34 |

(Special details of recruits were obtained for special cleaning assignments, and are not included in the estimate of complement required.)

## FACILITIES

*Location.*—The prosthetic facility should be located in the area in which the recruits in dental companies are to be barracked.

*Design.*—The over-all design of the prosthetic facility should permit the employment of Reserve prosthetic dental officers under conditions simulating those to which they have been accustomed in civilian practice. Since many of these officers are unable to carry out many of the procedures usually done by prosthodontists in the Navy during peacetime, a wartime prosthetic facility cannot be a mere enlargement of a regular Navy facility.

While it is most desirable to have the clinic and laboratory sections of a wartime prosthetic facility in the same building, the facility should be so designed that they can function and be administered as separate units. Personnel in the clinic section should not be permitted free access to the prosthetic laboratory, nor should laboratory personnel be permitted to enter the clinic section at will. Hence, separate locker facilities and entrances should be provided for the clinic and for the laboratory.

*Space.*—A complete technic which best fits the capabilities of the available personnel and the circumstances of the moment should be adopted; ample space and built-in features in office and laboratory should be provided where required to best accomplish each step in the technic from impression to finished case.

*Minimum space and lay-out requirements.*—Blueprints can be obtained of various large naval prosthetic facilities constructed during

World War II, and the most desirable features of each adapted to the needs of a particular activity. However, the need and value of certain features which are considered to be minimum requirements may not be apparent from the blueprints or recognized during the planning stage, and are therefore mentioned.

*Clinic Section*

(*a*) The waiting room should be large enough to seat a full company of men or such fraction of a company as it is planned to complete daily; adequate space must be provided for hanging their coats.

(*b*) Four-chair clinics should be adopted in preference to individual offices, thus saving construction costs and materials and permitting cooperative effort of the prosthetic dental officers.

(*c*) If four-chair clinics are utilized, two or three prosthetic offices should be provided where special technics required for unusually difficult cases may be employed and crown and bridge work performed.

(*d*) Each prosthetic dental officer should be provided with a suitable work table and bunsen burner, near the chair, where he can make necessary minor alterations to setups.

(*e*) A wall cabinet for the storage of cases in different stages of completion as they come from the laboratory should be provided for each dental officer. Cabinet design should be such as to permit hanging impression trays in a systematic manner.

(*f*) A separate telephone switchboard should be provided in all large activities and ready access to a telephone provided for each dental officer for communication with the laboratory.

(*g*) An auxiliary laboratory, with dental lathe and polishing equipment, should be readily accessible to the dental officers for making adjustments.

(*h*) A research room should be provided, if possible, where new procedures can be developed and new materials fitted to the adopted technic prior to their introduction in the laboratory.

(*i*) Adequate bulk storage space should be provided for the large amount of bulky supplies and stores necessary for continuous operation. Separate supply issue rooms should be provided for both the laboratory and clinic section.

(*j*) Separate facilities for examination and diagnosis should be provided in the same building if space is not available in a building already constructed. This should include a waiting room with a separate entrance, having seating capacity for a full company, space for three dental chairs, and the necessary equipment for two officers to diagnose cases and accomplish exploratory operative procedures when necessary.

869434°—50——7

(*k*) A lecture room, with blackboard, should be provided for the instruction of both dental officers and technicians.

(*l*) A suitable staff conference room is essential.

(*m*) A small maintenance room should be provided in which are stored the parts and tools necessary for maintaining all prosthetic equipment in proper condition, and for making minor repairs that do not warrant the services of the station's maintenance department.

(*n*) Separate head facilities for patients should be provided directly off the waiting room.

(*o*) Adequate clerical and administrative space should be provided.

(*p*) Provision should be made for photographing cases for record and instruction purposes.

(*q*) Space for darkroom, sleeping accommodations for the duty officer, heads, cleaning gear, slop sinks, and so forth, is also necessary.

*Prosthetic Laboratory*

(*a*) The most desirable prosthetic laboratory is one in which all full and partial denture procedures can be performed in one large room without any obstructions such as pillars. The ceiling should be higher than that of the clinic section, and there should be as many windows as possible. This will provide unobstructed visibility throughout the laboratory as well as adequate lighting and sufficient ventilation for dissipation of the heat from the numerous bunsen burners and other heating equipment.

(*b*) The ceiling should be soundproofed.

(*c*) Space should be provided for work benches for two dental officers at the end of the laboratory where cases enter and leave the laboratory. All impressions entering and work leaving the laboratory should be inspected by these officers. Only from their point of unobstructed view can the laboratory best be administered.

(*d*) Contrary to general belief, dental technicians' sit-down benches do not have to be placed next to windows for lighting purposes. Most technical work is done under artificial lighting and can be properly accomplished without eye strain under such lighting. Therefore, where the size and shape of the laboratory permits, it is most desirable to place the sit-down benches in even rows near the center of the room, and the required stand-up benches, curing pots, and polishing machines around the walls.

(*e*) The sit-down benches should be 30 to 32 inches high, and have a working surface 18 inches deep and 42 inches long. An 8-inch shelf approximately 7 inches above the working surface should be provided across the back of the working surface for case storage.

(*f*) The aisle between the sit-down benches should be 5 feet wide when technicians are sitting back to back, and 3 feet wide when all benches are facing in the same direction.

(*g*) The working surface of the stand-up benches should be 36½ inches from the floor, should be 30 inches deep, and as long as necessary. The stand-up bench should have a back shelf 6¼ inches high and 6 inches deep and have a 3½-inch recessed toe space at the floor. Also, adequate storage space for articulators, and so forth, should be provided.

(*h*) Sufficient sinks should be placed in positions immediately accessible to each technician.

(*i*) The arrangement of the sit-down benches should be such as to permit flexibility in their assignment to different procedures.

(*j*) The sections performing each procedure should be so located that the work may progress through the laboratory without backtracking.

(*k*) Sit-down and stand-up benches are carried on the Medical Supply Table. The stand-up benches are provided as separate units; each unit is designed for the purpose for which it is intended. Sufficient stand-up benches of appropriate design should be placed so that they are handy to the technicians using them. The amount and type of stand-up bench space required in each section of a laboratory designed to complete 200 dentures per day is shown in figure 1. A laboratory designed for a smaller work load can be assumed to have approximately the same proportion of sit-down and stand-up bench space, providing a simple prosthetic procedure is employed throughout. An accurate plan of bench arrangement can be obtained only by determining the exact dimensions of the benches currently on the Medical Supply Table and fitting them into a detailed scale drawing. The benches on the Medical Supply Table during World War II were superior in quality and cheaper than those that could have been constructed locally.

(*l*) In the interest of the technicians' health, suction systems should be installed in sit-down benches used by the gold, setup, and wax and rough-finish technicians. This system should have removable central filters so that all gold grindings can be reclaimed. This is most important and will result, at large activities, in the return of thousands of dollars to the government.

(*m*) Laboratory dental engines are not necessary; practically all prosthetic technicians working in civilian laboratories (they will make up a large part of the prosthetic technician complement)—are accustomed to and more adept at using the dental lathe to carry out all laboratory procedures. Also, it was conclusively shown during World War II that all technicians can learn to do all procedures better and faster with a dental lathe.

(*n*) There should be a supply room and supply and tooth counter, at one end of the laboratory, at which technicians can readily obtain

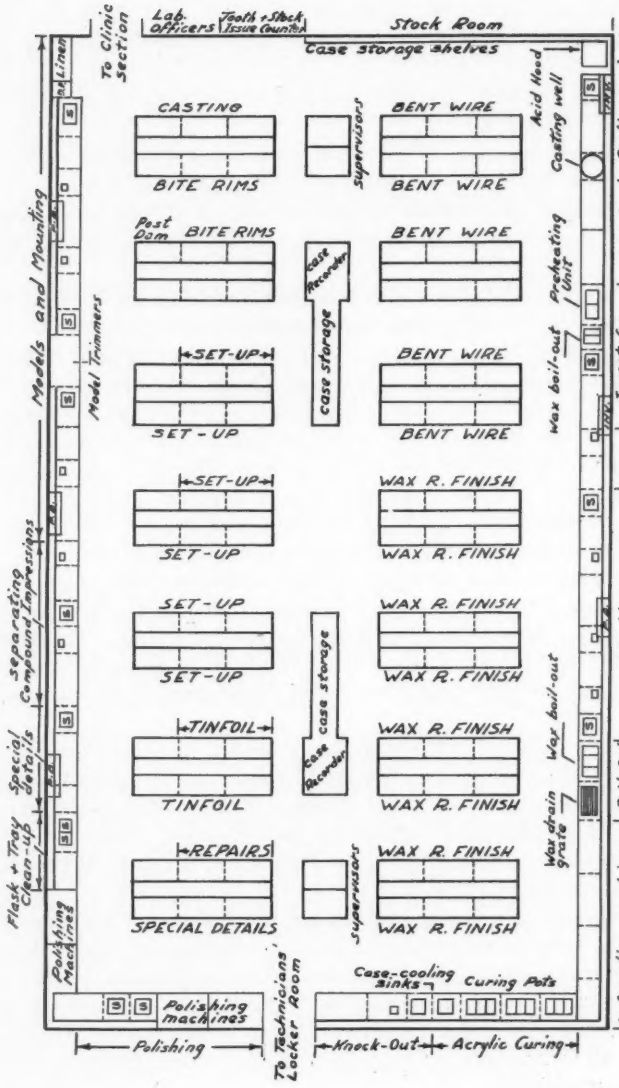



Figure 1.—Laboratory layout.

supplies as needed. A large vault or safe should be provided in conjunction with the supply room to store a sufficient amount of gold and teeth.

(*o*) Adequate shelving space must be provided for cases in case boxes. The amount of shelving required can be determined from the number of cases the laboratory is designed to complete per day and the number of days that will be required to complete each case. This shelving should be located as near the center of the laboratory as possible, divided, and clearly marked into sections for each full procedure to be done.

(*p*) In large laboratories a desk for a case recorder should be provided at each end of the case-storage shelves.

(*q*) A large double-section sink where impression trays and articulators can be cleaned and flasks alternately soaked and cleaned should be provided.

(*r*) If the construction of bent-wire skeletons is to be the general rule, a large preheating unit should be installed in the stand-up bench designated for soldering. A boil-out pot should be placed in the same bench.

(*s*) Adequate provision for heat-treating the volume of gold skeletons anticipated should be made.

(*t*) Benches should be provided in the center aisle of the laboratory for supervisors and inspectors.

(*u*) A lock should be provided on at least one drawer of each sit-down bench so technicians will have a means of preventing the loss of instruments.

(*v*) An extra gas outlet with a needle valve to provide a small flame for smoothing wax should be provided at sit-down benches.

(*w*) To promote a high degree of efficiency, recorded music should be played through a loud-speaker system.

## PROSTHETIC TECHNICIANS' SCHOOL

Experience has shown that because of the activity and noise it is undesirable to provide benches in a main prosthetic laboratory for the instruction of new technicians. Therefore, if a technicians' school should be included in the plans of a proposed prosthetic facility it should be considered as a separate unit and housed in a separate section of the building.

## TREATMENT PLAN

*Dental companies.*—During World War II the period of recruit training per company varied with the personnel requirements of the fleet, and, at times, was as little as 4 weeks. The prosthetic department may be ordered to provide the necessary prosthetic treatment for

each recruit during his period of training, regardless of how short the training period may be. Should that be the case, it may be impossible to accomplish the necessary operative treatment and extractions in time to allow sufficient healing so that suitable prosthetic treatment can be rendered during the regular training period. Another undesirable situation exists when prosthodontia patients are scattered throughout the companies in the training center. Where such is the case, training "musts" will interfere with the ability to provide special diet and treatments for patients; and the greatly increased dental clerical work required to keep track of the treatment for each recruit will become an insurmountable obstacle to the efficient administration of the prosthetic activity.

Under the previously mentioned circumstances it will be impossible to provide prosthetic restorations of the desired quality in the time allowed. Any attempt to transfer incompleted cases to companies graduating at a later date will result in the retention of an unpermissible number of recruits in training status.

The most satisfactory way to overcome all these obstacles and yet provide suitable prosthetic treatment is to (*a*) place all recruits in need of such treatment in special dental companies immediately upon induction; (*b*) stabilize the training period of such companies at a minimum of 8 weeks; (*c*) barrack all dental companies in the same regiment, in the area in which the prosthetic facility is located; (*d*) maintain by companies a record of all treatment required and completed; (*e*) schedule all treatment under a treatment coordinator; and (*f*) eliminate any conflict between training "musts" and required dental appointments through cooperation with the recruit training officer.

*Original examinations.*—It is assumed that in every large training center facilities will be provided for physical examination of all new recruits before they are assigned to a company for training. Dental officers conducting routine dental examinations of new recruits should be instructed to have all those who may require prosthesis reexamined by a prosthetic dental officer who will (*a*) definitely determine the need for prosthesis; (*b*) record the types of prostheses required; (*c*) assure that recruits in need of prostheses are placed in dental companies; and (*d*) send a list of all recruits assigned to a given dental company with the number and types of prostheses required, to a treatment coordinator in the main prosthetic facility.

*Treatment coordinator.*—A treatment coordinator is necessary to assure (*a*) that all the oral surgery and operative treatment required by a company is completed as soon as possible, and by a definite specified time to permit maximum healing; (*b*) that all prosthetic treatment is completed sufficiently in advance of the graduation date to permit

two adjustments on each restoration; and (*c*) that training periods and dental appointments of recruits do not conflict. The established deadline by which all oral surgery had to be completed at the U. S. Naval Training Center, Great Lakes, Ill., was the fifth day after a company's formation; the training period for dental companies was 8 weeks.

Before the treatment coordinator can accomplish these objectives, he must have an accurate record of the amount of various types of treatment necessary, and accordingly appoint recruits for that treatment. This cannot be done without a more accurate determination of the treatment requirements than is possible in the limited time allowed during the screening examinations. Therefore, the treatment coordinator must see that each company is reappointed as soon as possible for a thorough examination and final diagnosis.

*Final diagnosis.*—The entire dental company is reexamined, and a careful and as nearly final diagnosis as possible is made. To accomplish this (*a*) X-rays are taken whenever indicated; (*b*) questionable cavities are excavated to determine whether extraction is required, since the type of prosthetic treatment rendered may depend upon whether questionable teeth have to be extracted; and (*c*) a record of all extractions, surgical preparations, restorations, and the number and types of prosthetic replacements for each patient is forwarded to the treatment coordinator.

After the treatment requirements for a company have been determined, the treatment coordinator will advise each department of the amount of work to be accomplished and will work out a schedule of appointments with the head of each department and with the recruit training officer to assure that it is accomplished according to a set schedule.

*Oral surgery.*—A separate oral surgery department with a dental sick bay should be provided to treat men in dental companies; this *must* be under the direct control of the officer in charge of the prosthetic activity. This will (*a*) permit understanding and cooperation between the oral surgeon and the prosthodontist which is essential to good dentistry; (*b*) permit the prosthetic department to follow through on the diagnosis for prosthetic treatment without changes being made by oral surgeons unfamiliar with the requirements of prosthetic dentistry; (*c*) allow the prosthetic department control over oral surgical preparation and surgical procedures; and (*d*) permit the best possible cooperation with the prosthetic department in determining the desirability of rendering periodontia treatment, especially where teeth intended for abutments are concerned.

*Operative dentistry.*—A separate operative dentistry department should also be provided as a part of the prosthetic activity to assure that (*a*) the prosthetic department obtains the type of restorations

desired, and in which the specified relief for clasp rests has been provided; (*b*) fillings are finished and polished, so that the most desirable partial dentures can be provided; (*c*) contouring of restorations can be obtained when required; (*d*) the prosthetic department can be certain that large cavities will be filled when possible and desirable, and that the final diagnosis and healing time is not altered through the extraction of such teeth; (*e*) a prophylaxis is rendered where required, and each patient properly instructed in oral hygiene so that he is impressed with the fact that the success of the treatment depends upon his personal efforts; (*f*) all operative treatment requirements are completed according to a predetermined schedule; and (*g*) patients not in dental companies are not assigned to this facility for treatment and thus the schedule of treatment for dental companies is not disrupted.

A company roster listing operative restorations required is forwarded to the operative dentistry department by the treatment coordinator, and a deadline set for completion of restorations for the entire company. Although the deadline can be set much later in the training-period, this phase of the treatment should be completed as soon as possible so that sufficient healing time may still remain if additional extractions are required.

*Examination prior to prosthesis.*—On approximately the fifteenth day prior to graduation, a company in which all preprosthetic treatment has been completed is reexamined; recruits whose mouths will not be in condition for impressions on the ninth day prior to graduation are transferred to a company graduating at a later date. The number of such transfers at the U. S. Naval Training Center, Great Lakes, Ill., had to be kept at a minimum, for two reasons: (*a*) Recruit Training considered it an inexcusable waste of manpower to retain any recruits beyond their established training period; and (*b*) frequently there was sufficient barracks space to barrack only a very small number of hold-overs. Therefore, it was obvious that a schedule of the treatments had to be established and rigidly adhered to if maximum healing time was to be provided and suitable prosthetic treatment rendered by the end of the 8-week training period.

## PROSTHETIC DEPARTMENT

*Records.*—A company roster and record of the prosthetic requirements of each recruit is furnished to the prosthetic department by the treatment coordinator as soon as possible after final diagnosis has been made, so that the prosthetic department may know in advance the types and amount of prosthetic treatment that must be rendered each day. Final corrections to this record are made at the time of examination for prosthesis. Two file cards are then made out for each patient, one for the clinic and one for the laboratory, and filed

on separate file panels identified by the company number. A list of the procedures to be accomplished by the clinic and by the laboratory are placed, as column headings, across the top of the respective panels. The panels are retained at the record desk (where impressions are received and a case number assigned before they are sent into the laboratory) until a case number has been assigned to each patient listed on the panel, and the number recorded on his card on each panel. The laboratory panel is then sent to the first case recorder. Clinic and laboratory procedures are checked off each patient's card as they are completed.

*Scheduling.*—The required prosthetic treatment is accomplished by companies, insofar as possible, and is begun 9 days before graduation. This permits the maximum healing time prior to impressions, and still allows time for adjustments and possible make-overs. Each prosthetic procedure is accomplished according to a definite schedule and completed by a definite time, both by the dental officers and by the various laboratory sections.

Each dental company is divided in two sections for better scheduling various clinic treatment procedures, and to prevent the overloading of any portion of the laboratory at any time.

Schedules which proved satisfactory are as follows:

CLINIC SCHEDULE

| *Section I* | *Section II* |
|---|---|
| Chairs 7 to 14 and 21 to 28 inclusive: | Chairs 1 to 6 and 15 to 20 inclusive: |
| 0800 to 1000 Impressions. | 0800 to 0930 Bites. |
| 1000 to 1130 Adjustments. | 0930 to 1130 Impressions. |
| 1230 to 1330 Try-ins. | 1230 to 1400 Adjustments. |
| 1330 to 1500 Insertions. | 1400 to 1500 Try-ins. |
| 1500 to 1630 Bites. | 1500 to 1630 Insertions. |

Each patient is assigned to the same dental officer for the completion of all required procedures.

TABLE 1.—*Laboratory schedule.*

| | Section | |
|---|---|---|
| | 1 | 2 |
| Models made | Prior to 1230 first day | Prior to 1400 first day. |
| Base plates and biterims | Prior to 1500 first day | Prior to 1630 first day. |
| Bites must be back in laboratory | Prior to 1630 first day | Prior to 1000 second day. |
| Cases mounted | Prior to 1230 second day | Prior to 1500 second day. |
| Bent wire | Prior to 1630 second day | Prior to 1230 third day. |
| Set up | Prior to 1230 third day | Prior to 1400 cases requiring try-in third day, 1630 all other cases. |
| Try-in in laboratory | Prior to 1400 third day | Prior to 1500 third day. |
| Wax-up | Prior to 1230 fourth day | Prior to 1500 fourth day. |
| Invest process | Prior to 1630 fourth day | Prior to 1100 fifth day. |
| Finish | Prior to 1230 sixth day | Prior to 1500 sixth day. |

These schedules may be altered according to the technic employed. Details of organization and administration necessary to maintain a certain schedule must be determined but this will not be discussed.

The rendering of prosthetic treatment by full or half companies assures an even schedule and flow of work through the clinic and laboratory from day to day. Since the rate of induction is not constant, steps must be taken to assure that nearly the same number of men be given prosthetic treatment daily. This may be done as follows:

(*a*) Vary the standards for those who require prosthetic treatment.

1. Set a minimum standard of, for example, the equivalent of three bicuspids in occlusion, and do not place recruits meeting this standard in dental companies when the induction rate is so heavy that more than one prosthetic dental company would be formed per day if this minimum standard were not in effect.
2. Liberalize the minimum standard during slack periods of induction when a company a day would not otherwise be formed. If additional prosthetic treatment can be completed it will relieve the work load at activities to which the recruit might be sent.

(*b*) Vary the amount of prosthetic treatment rendered, within the minimum and maximum limits, as necessary to form one company a day 5 days per week. Reserve Saturdays for the treatment of station personnel and for the instruction of dental officers and corpsmen.

(*c*) Appoint companies for prosthetic treatment on the basis of one-half a company per day when the induction is extremely slow, and make sure that the prosthetic treatment needs of all station personnel are treated to the fullest extent possible during the slack period.

(*d*) Work on a full schedule of a company a day on Saturdays and Sundays when necessary, and eliminate all treatment of station personnel during this period.

During slack periods, the utilization of the services of personnel assigned to the prosthetic department's oral surgery and operative clinics—elsewhere and on a "loan" basis—does not present any special problem; their services can be employed in meeting the dental treatment requirements of recruits not in dental companies. The services of the prosthetic dental officers, however, cannot be so employed without a waste of man-hours on the part of the prosthetic laboratory technicians. This is another reason for maintaining as even a flow of prosthetic work as possible.

## SELECTION OF PROSTHETIC TECHNIC

The prosthetic technic employed should be one that will provide the best possible prosthetic restorations, and one that can be done using the

equipment and materials listed in the Supply Catalogue. The technic shall be determined by the dental officer in charge. It is quite possible that circumstances will not permit the use of a technic which is too elaborate to assure the quality of prosthesis which may be done by prosthetic specialists under more ideal conditions. It may, for example, be impossible to routinely obtain Gothic arch tracings or make use of the face-bow because of the unfamiliarity of the employed dental officers with such devices and other personnel and time factors. It may be possible to fabricate only the simplest types of bent-wire partials because of the limited capabilities of the technicians. However, the best possible technic should be devised and employed, and a constant endeavor made to improve it.

## LABORATORY CASE ROUTINE

A complete systematic laboratory routine must be worked out with provision for determining the exact stage of completion and location of the case for any patient at any time.

In order to insure the highest quality of workmanship, it is essential that all impressions be inspected upon entering the laboratory by the laboratory officer or his assistant. It is also essential that the work completed by each technician be supervised and inspected.

As an aid in identifying the large number of cases in a large laboratory, the assigned case number should be inscribed as soon as possible in the models for each case. Technicians should draw cases from a centrally located case recorder who records the bench number of the technician opposite the patient's name on the laboratory file panel. In addition, prior to flasking, it is advisable to make for each case, an occlusal splint which can be kept in the case box and used for positive identification and for determining any alterations in occlusion when the case is completed.

### *Average time required for laboratory procedures*

*Models.*—Pouring, 8 minutes. Trimming, 2 minutes. Total time for completion, 45 minutes.

*Outline and postdamming.*—Full upper model, 5 minutes. (31.3 percent of all cases are full uppers. Therefore, on basis of 200 dentures per day, $200 \times 31.3 = 63$ full uppers.)

*Baseplates and biterims.*—15 minutes to make.

*Mounting cases.*—Upper and lower models on articulator, 3 minutes.

*Bent-wire skeletons.*—Lowers, 1 hour, 26 minutes; uppers, 1 hour, 6 minutes.

*Set-ups.*—32 minutes per case.

*Waxing.*—25 minutes per case.

*Occlusal matrix.*—The occlusal matrix is made for 12 cases at a time. Average time for the 12 cases, 10 minutes.

*Investing lower half.*—8 cases are invested at a time (2 men). Average time required, 10 minutes.

*Tinfoiling.*—9 minutes per case.

*Investing upper half.*—8 cases are invested at a time (2 men). Average time required, 10 minutes.

*Stone core for upper half of flask.*—12 cases are poured at a time. Average time required, 17 minutes.

*Boil-out.*—12 cases are boiled out at a time. Average time required, 10 minutes.

*Packing.*—12 cases are packed at a time (2 men). Average time required, 25 minutes.

*Processing.*—12 cases are processed at a time. Average time required, 2 hours.

*Knock-out.*—5 minutes per case.

*Rough finish.*—25 minutes per case.

*Polishing.*—13 minutes per case.

*Average total time required to complete a full denture.*—6 hours 19 minutes (includes 45 minutes cooling time).

*Average total time required to complete a lower partial denture.*—7 hours 40 minutes (includes 45 minutes cooling time).

*Average total time required to complete an upper partial denture.*—7 hours 20 minutes (45 minutes cooling time added).

Twenty-four percent of all cases are partial uppers, 31.8 percent of all cases are partial lowers. On basis of 200 dentures per day: $200 \times 24 = 48$ partial uppers per day; $200 \times 31.8 = 64$ partial lowers per day.

## MISCELLANEOUS OBSERVATIONS

*Navy prosthetic officer should be appointed.*—Cooperation in rendering of prosthetic treatment during wartime must extend beyond individual activities. A prosthetic officer should be appointed to correlate the prosthetic effort of all prosthetic activities for the good of the entire service.

*Cooperation with other dental activities.*—Cooperation with station dental activities outside the prosthetic department is essential. The interchanging of dental officers and technicians between the prosthetic department and other dental activities of the station will permit the assignment of dental officers according to the predetermined work load. Also, with the prosthetic department working on a definite schedule, it is essential that when necessary, additional men with proper qualifications, from other station activities, be assigned to each department connected with the prosthetic effort.

*Dental officers should not be permitted to enter laboratory.*—Dental officers assigned to a large naval prosthetic activity should not be permitted to enter the prosthetic laboratory at will, for they will

select technicians who best suit their personal preferences to work on their cases, or insist on performing laboratory work themselves which can be accomplished much better and faster by a competent technician.

*Standardized technic must be employed.*—A standard technic must be worked out for both the clinic and laboratory and must be employed by everyone concerned, so that equipment and supplies will not be wasted and administrative details kept as simple as possible. Should a change in the established technic appear advisable it should be adopted, after its real worth has been determined, by every dental officer and technician concerned.

*Prosthetic technicians' capabilities must be used to best advantage.*—While it has been the Navy's policy to insist that prosthetic technicians be trained to perform all laboratory procedures, this policy is not always feasible during wartime. Technicians may have to be trained in a limited number of procedures at which they are most adept and assigned accordingly.

*Allocation of ratings to civilian prosthetic technicians upon induction.*—To insure maximum efficiency and unimpaired morale, no former civilian laboratory worker should be rated merely on the basis of his previous laboratory service or his employer's letter of recommendation. His qualifications for rating should be determined accurately by a practical examination.

*Prosthetic technicians should be responsible for work accomplished.*—High quality workmanship by all laboratory technicians must be assured to minimize the number of cases returned to the laboratory for correction or remake. Each technician should be made responsible for a complete procedure so that responsibility can be fairly placed. Responsibility cannot be as easily fixed if a laboratory operates on a shift basis.

*Uniform of enlisted personnel should be distinctive.*—Dental laboratory technicians should wear identifying working uniforms different from those in the clinic section, to simplify the administrative control of the large number of enlisted personnel in one building.

*All completed cases should be inspected.*—All completed prosthetic cases after oral insertion must be inspected by a qualified prosthetic dental officer to assure that the best possible prosthetic treatment has been rendered each patient.

# About the Army Medical Department

## I. Emergency Personnel Planning

PAUL I. ROBINSON, *Colonel, MC, U. S. A.*[1]

EMERGENCIES in personnel requirements for medical officers for special boards, special commissions, maneuvers, and many other details frequently tax the Personnel Division to the utmost. Every effort is made to consider the personal problems of individual officers. In a recent maneuver that required 33 medical officers, 7 had to be replaced for such reasons as personal problems of a legal and domestic nature, or illness of other officers in the installation where the officer was assigned. These requirements are met with the least possible disruption to the medical installation concerned.

One of our most difficult problems will be the acute shortage of medical officers this summer in the overseas commands. The shortage will exist for only about 3 months. It results from the fact that almost 500 young medical officers terminate their tours of duty in May and June and replacements from the military and civilian residency and internship programs cannot be made available to the overseas commands before August. It is necessary, therefore, that representations be made to the Department of the Army General Staff, that certain procurement programs be established, certain training programs must be temporarily interrupted, and everyone be kept informed. Typical of the early steps to meet this problem is the personal letter of information sent to the surgeons of major overseas commands. Such a letter is paraphrased because it illustrates many of the steps that are routinely taken in personnel work.

As you know, we have been carefully studying the situation with regard to medical officers in your command because of the fact that you will lose so many officers in May and June. This is a preliminary letter of information to acquaint you with the efforts that we are making. The Surgeon General has agreed that we take 100 residents out of the teaching hospitals in the United States and

[1] Personnel Division, Office of the Surgeon General.

send them on temporary duty from 15 May to 15 August, distributed among all the overseas commands. The proposal of taking the 100 residents was sent to the Deputy Chief of Staff, Personnel and Administration Division, who thought our efforts so far were laudable but that they were not sufficient.

Other efforts which presently are being made to obtain more medical officers for 3 months' duty are as follows: (a) A study is in progress to determine whether more residents can be taken for this temporary duty. (b) The Navy has been asked to lend us 50 doctors for this period and is conscientiously studying the problem. (c) The First, Second, and Sixth Army Areas will be asked to release about 35 doctors from their areas for temporary duty during this period. (d) Efforts may be made through publicity to obtain as many Reserve officers on active duty as possible for this period. (e) Investigation will be made into employing civilian doctors to cover this interval.

It is possible that before you receive this letter, an official TWX will have been sent from the Department of the Army to your command asking air priority for some or all of the transportation which would be involved covering the doctors who will be gained by the above processes. If so, I hope you will use your influence to arrange the necessary air priority because otherwise we would not be able to furnish medical officers for regular assignment to replace those you will lose until about 15 August. This letter is purely informative. We will notify you officially of the actions which are approved and placed into effect as soon as possible.

---

## II. Career Planning

ANNA E. CAREY[1]

ALTHOUGH the Career Planning Program for the Army was established in June 1947, the Surgeon General in December 1945 took the first major step in what is now a well-organized Career Guidance Program. At that time a memorandum was sent to the Chief of Staff, U. S. Army, requesting his assistance in securing the immediate release of 71 specially selected officers on duty in overseas theaters and with major forces in the United States, for the purpose of retraining them professionally. The termination of the war in August 1945 found practically all our physically fit medical officers of the Regular Army under 56 years of age on administrative or tactical duties, many overseas; and while they had had no opportunity to share in the teaching or guidance with the many

[1] Personnel Division, Office of the Surgeon General.

professional specialists and teachers who had come on active duty during the war (from our best medical colleges) they would soon be expected to replace them. With few exceptions these officers were given an intensive training program in either a military or civilian institution in the specialty in which they had previously shown an interest. Many have since been certified by the various specialty boards and are now the bulwark of the residency training program in Army hospitals.

At about the same time it became evident that because of general chaos in Europe the medical center of the world would shift to the United States, and that civilian medicine was making preparation to meet the demand for postgraduate training in medicine from the many (75 to 80 percent) young physicians leaving the Army. The high level of medicine practiced in the Army during the war was, in part, a spur to this ambition. It was realized that if the Army was to procure the 200 percent increase of Regular Army officers over the prewar authorization and continue to hold the best officers in the Corps, it had not only to maintain its high standard of practice but also to meet the highest standard of civilian medical training. The accepted medium for the determination of professional ability in civilian medicine was and is certification by an American specialty board.

Plans were then instituted whereby some of the general hospitals of the Army were to receive full accreditation by the recognized civilian medical agencies for intern and resident training in various specialties. Accreditation of Army hospitals is one of the most important steps made toward establishing the medical service of the Army on a par with leading universities and medical centers in the country. This approval came slowly at first but has increased in the past 4 years, and today the Army Residency Program is recognized as one of the best in the country.

Prior to beginning the program, a complete study of the whole Corps was made to ascertain its present and potential professional capabilities. Initial requirements were established and the total number of specialists required to staff the general and station hospitals that would be in operation in the Zone of the Interior and overseas theaters in 1946 and 1947 was ascertained. Continuous study of availabilities and requirements is made to meet the changing mission of the Army and to insure that each member of the Corps will have the opportunity to progress in his chosen specialty and to meet the needs of the Army.

These studies have been implemented by various means such as the career guidance ledger which contains the name of each Regular Army medical officer, listed chronologically according to date of retirement for age, in accordance with the primary MOS classification and

degree of ability in the classification. The purpose of the ledger is to obtain specialty counts of Regular Army medical officers, to serve as an instrument for classification control and to establish training requirements in specialties in accordance with the age groups of personnel in the various specialties. By means of the ledger and a residency space control book, the number of officers qualified and under training in a specialty is always available. This is the Medical Department guide for training officers required in various specialties to meet the needs of the Army.

A career card has been prepared and is kept current for each Regular Army medical officer. Professional progression patterns for the different professional specialties and for a career in command and staff positions have been completed. These patterns have been developed as a guide for the rotation of assignments of Regular Army medical officers, and are correlated with the needs of the Army and the desires and demonstrated ability of individual officers. At present 162 career patterns have been developed for medical officers, and copies mailed to them. Each officer will eventually receive one. These patterns show to what degree and positions an officer may advance in his specialty. These patterns have been received with enthusiasm and have stimulated the interest of junior officers in continuing a career in the Medical Corps.

The first year of formal residency training in an Army hospital started on 1 January 1947. Since then 700 medical officers have participated in the program. Of this number 163 have been assigned to duty after completion of the 1 to 3 years of formal training they required. Ninety officers have been withdrawn from the program prior to completion of training among whom were included officers assigned to civilian training (most of whom were also assigned as instructors in ROTC units), those who showed no aptitude in the chosen specialty, those who asked for release (some of whom resigned from the Army), and those who were forced out for lack of space under a pyramidal system. About 20 were withdrawn at the end of 1947 to go overseas to meet an urgent need. Some of these have already been returned to the program and all will have reentered by the end of 1950.

On 1 January 1950, there were 466 participants. Of these 388 are in the Army and 78 are in the Air Force. At present 275 officers are participating in the Civilian Residency Program. Of this number about 45 will complete their training in June 1950 and will then be assigned to duty in an Army installation. Twenty officers who participated in the program have already been assigned to duty in an Army installation, either by reason of having completed the required formal training or by request on the part of the officer.

869434°—50——8

All Regular Army Medical Department officers, with the exception of female officers and all Reserve and AUS officers, who have so requested, have been awarded a constructive credit equivalent, in accordance with War Department Circular No. 62, dated 6 March 1947. A board is in continuous session to accomplish this and to review reclaimers.

In addition to specialty training of Medical Department officers in civilian institutions, 60 Medical Department officers received training in public health, 18 in hospital administration, 4 in personnel management, and 4 in business administration within the past 4 years.

Since publication of TM 12–425A, July 1947, the Surgeon General has been authorized to classify all medical and dental officers in military occupational specialties throughout the Army. An annual reevaluation of all such officers is made on the basis of training and experience. Similar centralized control of the classification of all other corps of the Medical Department has been recommended but has not yet been published. New specialty codes, titles, and specifications are prepared when required to meet varying missions of the Army.

In 1945 there were 75 officers on duty who were certified by the American specialty boards. As of December 1949 there were 171 board members. The increase in certifications is greater than would at first appear since in the period from 1945 to date there were losses among those certified as follows:

| | | | |
|---|---|---|---|
| Retired | 25 | Resigned | 2 |
| Transferred to Air Force | 15 | Deceased | 2 |

The total number of Regular Army medical officers who are specialty board members is shown in table 1:

TABLE 1.

| *Board* | *Number* |
|---|---|
| Anesthesia | 2 |
| Dermatology and syphilology | 3 |
| Internal medicine | 45 |
| Internal medicine; neurology; preventive medicine and public health | 1 |
| Physical medicine | 2 |
| Psychiatry and neurology | 6 |
| Psychiatry | 5 |
| Neurosurgery | 0 |
| Obstetrics and gynecology | 3 |
| Ophthalmology and otolaryngology | 1 |
| Ophthalmology | 4 |
| Otolaryngology | 6 |
| Orthopedic surgery | 4 |
| Pathologic anatomy and clinical pathology | 8 |
| Pathologic anatomy | 12 |
| Clinical pathology | 3 |
| Pathologic anatomy; clinical pathology; preventive medicine and public health | 1 |

TABLE 1—Continued

| *Board* | *Number* |
|---|---|
| Preventive medicine and public health | 17 |
| Pediatrics | 4 |
| Plastic surgery | 1 |
| Radiology | 20 |
| General surgery | 16 |
| Thoracic surgery | 1 |
| Urology | 6 |
| Total | 171 |

For the Medical Service Corps a career pattern has been prepared to show the several specialty groups of the Corps, the type of activities to which the personnel of each group will be assigned, and the phases of their military training in service schools. General assignment instructions have also been formulated to prescribe how the careers of each group will be developed through formal training and duty assignments. These instructions indicate the order in which the various types of assignments and training will be received, during what period of service, and for how long. All of this material has been presented to the Army through TM 20–605, June 1948. Subsequent to this publication, individual patterns were prepared for each MOS in which a significant number of Medical Service Corps officers were engaged. For the scientific groups a chart showed how the assignments and training of officers holding a given MOS would progress from entry into the service until retirement. Normal job titles and installation types were shown, as were the types of courses and schools. For the administrative groups, each significant MOS was also included but more opportunity was presented for officers to become proficient in several specialties: for these officers the maximum versatility is desired.

With these individual patterns it became possible to plan the activities of officers for several years to come. Each officer's qualifications are studied in relation to the pattern appropriate to his MOS and an assignment and training plan is worked out to achieve the officer's maximum development. A valuable byproduct of this planning is the assistance it gives in having the proper officer available at the time vacancies in training or duty occur. A constant review is made of field reports to determine whether each officer's training is being used to proper advantage and whether he is measuring up to expectations. Changes in his assignment or his plan may be made based on reported use or performance.

For use in the control of assignments to various job positions and formal training, to prevent overtraining in some specialties and under-

training in others, a study was made of the T/O and E's and T/D's of all units and installations where Medical Service Corps officers are used and total requirements for each MOS were formulated. A file of all Medical Service Corps officers by MOS was set up as an inventory of the specialists on hand. This included a ledger similar to that previously described for medical officers. Career cards have been prepared for all Regular Army Medical Service Corps officers and are kept current.

To define more clearly the extent of training of officers in each type of specialty, eight new MOS's were prepared to cover the medical supply field. Alphabetical prefixes were prepared and submitted for publication to make the scientific MOS's more explicit. The letters D, C, and B, are awarded for progressive training and experience in the professional field and prefix A goes to officers who have distinguished themselves in the field after training has been completed. New MOS's will be prepared as required.

A basic career pattern has also been developed for the following corps: Dental, Veterinary, Army Nurse, and Women's Medical Specialist. Because of the limitation of these special fields rotation of assignments outside the field is not practicable. During the period of basic training rotation will be within the various types of duties in the field. Subsequent to classification in a specialized area of the field it is planned that an officer's assignments will continue in the area indicated.

In addition to continuing the study of the training and assignment of Medical Department officers as indicated above, it is planned in the next year to put more emphasis on training in military medicine. It is hoped to resume giving the basic course in the fall of 1950. This course will be divided into a medical and field phase. The medical phase will be given first to about 25 medical officers at the Army Medical Center, Washington, D. C. The field phase will follow, in January 1951 at the Brooke Army Medical Center, Fort Sam Houston, Tex. In addition to the 25 medical officers it is expected that 10 Dental Corps, 10 Veterinary Corps, and 25 Medical Service Corps officers will take the second phase. It is also expected that several officers of the Medical, Dental, Veterinary, and Medical Service Corps can be made available to take the advanced course at the Medical Field Service School each year from now on.

While the Career Management Program of the Army is designed only for Regular Army officers, in implementing it the Surgeon General is making provision for the maximum development and best use of Reserve officers on active duty. This is particularly true in the case of Medical Service Corps officers where the ratio is three Reserves to each Regular officer and where many of the officers wish to make a

career of the Army. The Career Guidance Section is alert to every possibility for training or experience that will render these officers better qualified for service with the Army.

Another function of the Career Guidance Section is to determine the best potential war assignment for all Regular Army officers of the Medical and Medical Service Corps, and to keep current such records for immediate use. Early in 1947 a file of principal war service administrative positions for Regular Army medical officers was developed. This information has been correlated with the records since the end of the war, with advancement by reason of military training or experience, and with professional training or experience, and a potential war assignment has been designated.

In October 1949 initial mobilization assignments were determined for about 900 Regular Army medical officers and 600 Medical Service Corps officers to fill key administrative and professional positions. These assignments are being reviewed periodically and changes will be made to meet adjustments deemed necessary by reason of promotion or changes in career patterns.

## The Control of Communicable Diseases

The Control of Communicable Diseases, 7th edition, American Public Health Association, was recently added to the List of Recommended Books for the Army Medical Department. One copy of this handbook will be issued to each medical officer. All installations and headquarters should obtain the necessary number of copies of this book in order that all medical officers may receive their copy at the earliest possible date. Additional copies are recommended for reference in headquarters offices and in medical libraries.

This new edition has been brought up to date by the Subcommittee on Communicable Disease Control of the Committee on Research and Standards of the American Public Health Association. The previous editions have long been accepted as an official statement of acceptable procedure by the Public Health Service. The present edition continues to carry this recognition and in addition has been accepted by several foreign countries. It has been approved in principle by the Surgeons General of the Army, Navy, and Air Force. As such it may be used for definition of accepted communicable disease control procedures where specific instructions are omitted in official Army publications.

Most medical officers believe they thoroughly understand the methods of controlling the spread of disease. Usually, however, when demands are made for this knowledge they find it to be vague and out of date. This fact is not recognized until the officer is suddenly called on to give his recommendations for the control of a disease encountered during military operation in an area foreign to him. When instances of this type occur a reference book is most welcome and often the least accessible. Since The Control of Communicable Diseases, is small in size, can be carried anywhere and gives all the salient information required in outline form, it readily fulfills the need in these unusual situations. The medical officer who is conscious of his preventive medicine responsibilities will have occasion to use the book almost daily whether he is assigned to a hospital, staff, or field position.

EDITOR'S NOTE.—The Bureau of Medicine and Surgery will distribute The Control of Communicable Diseases to all Medical Corps officers, Medical Service Corps officers, and to all ships and stations of the Navy Department.

THE PHYSICIAN MUST KNOW WHAT HIS PREDECESSORS HAVE KNOWN IF HE DOES NOT WISH TO DECEIVE BOTH HIMSELF AND OTHERS.—*Hippocrates.*

# EDITORIAL

## Clinical and Laboratory Use of Isotopes

At the present time radioactive isotopes are used to a greater extent in physiologic research than in therapy. The results of the therapeutic application of isotopes have been disappointing.

*Radioactive iodine* ($_{53}I^{131}$).—Radioactive isotopes of iodine are of value in the treatment of hyperthyroidism. About 80 percent of the patients with thyroids 60 to 75 gm. in size, treated with the appropriate dose of radioactive iodine, have been cured. Myxedema occasionally occurs.

Radioactive iodine is also of value in the relief of pain in angina pectoris and congestive heart failure; by lessening thyroid activity and diminishing the body's demand upon the heart the distress and pain of angina pectoris and the dyspnea of congestive heart failure is alleviated to an extent that enables the patient to resume physical activity.

Carcinoma of the thyroid does not respond to treatment with radioactive iodine because hyperthyroidism is not a clinical feature and the malignant thyroid tissue does not take up radioactive iodine. In only a small percent of carcinomas of the thyroid is the tumor capable of taking up and storing iodine. In some instances the metastatic lesion produces true hyperthyroidism and the uptake of radioactive iodine in such instances is the same as in toxic goiter.

*Radioactive iron* ($Fe^{59}$).—With the use of this isotope it has been shown that young red cells are more sensitive to hypotonic solutions than are mature red cells; that plasmodia prevails in young red cells; and that Nembutal will drive red blood cells into the spleen and therefore the administration of Nembutal in shock is dangerous. Further, that in the refrigeration of red blood cells, the addition of dextrose to the citrate anticoagulant and the maintenance of a slightly acid reaction of the diluted plasma and an optimal dilution is essential to prolonged preservation of red blood cells. In certain acid citrate solutions whole

blood can be safely preserved with up to 70 percent of viability for 3 weeks. Cells preserved by the addition of citrate alone are almost entirely broken down within 24 hours in the new host, after only 2 weeks of preservation in vitro. Transfused red blood cells live for 3 weeks in the recipient.

Further study indicates that iron, once admitted to the body, is stored in combination with protein and is not excreted, and is only slightly absorbed except in iron deficiency anemia; that anemia of infection is due to the inability of the bone marrow to utilize iron; that the body loses only a very small fraction of iron and the daily requirement is only about 0.001 to 0.002 gm. since about 90 to 95 percent of the iron in the red cell is reutilized when the red cell dies; that therapeutic administration of iron is necessary for donors, particularly for those who are repeated donors; that blood transfusion is the most effective means of administering iron; that the new red cell in the bone marrow can assimilate iron for a few days, but the mature red cell cannot; that folic acid and liver extract increase the ability of young red cells to take up iron; and that iron is used in large quantities for muscle hemoglobin and is an important constituent not only of the red blood cells but also of cytochrome.

Lysine is utilized by the body to form other amino acids with which to build proteins of blood plasma and red blood cells.

*Radioactive zinc* ($_{30}Zn^{65}$).—With this isotope it has been demonstrated that zinc plays an important part in the life cycle of the white blood cell and that in leukemia, the white blood cells show a pronounced zinc deficiency.

*Radioactive phosphorus* ($_{15}P^{32}$).—When given parenterally, radioactive phosphorus is concentrated in rapidly growing tissue, particularly in the nucleo-protein fraction and in bone.

In the treatment of polycythemia vera, radioactive phosphorus (disodium phosphate) given either orally or parenterally, is, at the present time, preferred treatment. Lawrence treated 30 patients with one course consisting of 2 injections of 3 to 6 milliecuries of radioactive phosphorus on the average of once every 3 years; 17 percent remained normal for 3 years after one course of treatment. Some have not needed retreatment for as long as 8 years.

Low-Beer treats radiosensitive superficial skin lesions by soaking a small piece of blotting paper in a labeled radioactive phosphate solution and applying this to the lesion. The beta rays from $P^{32}$ have a "half-penetration" of about 1 millimeter in the tissues. It is possible that $P^{32}$ may prove superior to high voltage roentgen therapy or radium in the treatment of such lesions. In suspected breast lesions Low-Beer and his associates have administered radioactive phosphorus and then, by surveying both breasts with a Strajman Counter, they found that if the suspected area has a count of 25 percent above

that of the normal breast, and if a breast abscess can be ruled out, carcinoma can be diagnosed. This is applicable, of course, only to superficially located malignant lesions. It does not apply to slowly growing mucoid carcinoma.

A similar technique can also be employed to locate metastatic lesions and to diagnose mycosis fungoides, dermal carcinoma, and cutaneous Hodgkin's disease. Multiple myeloma can be differentiated from carcinoma by the administration of $P^{32}$; multiple myeloma does not absorb the isotope.

$P^{32}$ can be used to locate brain tumors during surgery. These tumors absorb many times as much $P^{32}$ as does the normal brain tissue, in some cases over 100 times as much; however, the rays penetrate only a little more than one-fourth inch through the brain tissue and a specially devised Gieger-Mueller counter, of a diameter comparable to the size of a ventricular needle, is employed to locate the brain tumor.

*Radioactive sodium* ($_{11}Na^{24}$).—Radioactive sodium can be used to evaluate the circulation in such conditions as trench and immersion foot, and the blood supply of an extremity prior to amputation. Vascularity is measured by determining the distribution of radioactive sodium in the blood and the extravascular tissue fluids of the part.

BIBLIOGRAPHY

1. Atomic Energy and the Life Sciences. United States Atomic Energy Commission, Washington, D. C., July 1949.
2. DOUGHERTY, E. C., and LAWRENCE, J. H.: Medical progress: isotopes in clinical and experimental medicine. California Med. **69**: 58–78, July 1948.
3. DOUGHERTY, E. C., and LAWRENCE, J. H.: Medical progress: isotopes in clinical and experimental medicine. California Med. **69**: 148–153, Aug. 1948.
4. SEARS, T. P., and ALLEN, K. D. A.: Isotopes and the new alchemy. Rocky Mountain M. J. **46**: 931–936, Nov. 1949.

—J. L. S.

# BOOK REVIEWS AND BOOKS RECEIVED

Publishers submitting books for review are requested to address them as follows:

The Editor,
UNITED STATES ARMED FORCES MEDICAL JOURNAL,
Bureau of Medicine and Surgery, Navy Department,
Washington 25, D. C.

(For review)

---

SENSORY MECHANISMS OF THE RETINA, with an appendix on Electroretinography, by Ragnar Granit, M. D., *Director of the Nobel Institute for Neurophysiology, Professor of Neurophysiology, The Royal Caroline Institute, Stockholm.* 412 pages, illustrated. Oxford University Press, New York, N. Y., publishers, 1947. Price $11.

This book primarily deals with the transformation of the study of the electrical responses of the eye into a scientific method capable of analyzing the intricate sensory mechanism of the retina. It is divided into four sections: (*a*) The Electrical Signs of Excitation and Inhibition of the Retina and Optic Nerve; (*b*) The Properties of Rods and Cones. Variations in Area, Intensity and Duration of the Stimulus in Dark and Light Adaptation; (*c*) The Properties of the Photosensitive Substances and the Mechanism of Excitation; and (*d*) Colour Reception.

Electrophysiology of the retina dates from the middle of the last century when DuBois Reymond's discovery of the negative variation in nerves and the resting potential of the eye (1849) prompted Holmgren to apply electrodes to the eye. His galvanometer gave a deflection when the eye was illuminated and another when the light was turned off. He thereupon described what is now known as the electroretinogram in a paper published in 1865. Since then knowledge in this field has proceeded hand in hand with electrophysiology in general. The development of valve amplification has enabled electrophysiology to make great strides in the last two decades. The electroretinogram is a composite curve which integrates the complex electrical responses to stimulation in the retina. Granit's great contribution to the physiology of vision has been the revelation of the processes in the retina and optic nerve underlying not only scotopic but also photopic vision. He has shown that the retinogram is the only measurable index of what takes place in the retina in response to changes in illumination. He has found two chief types of electroretinograms: the E-Retinogram which is dominated by rods; and the I-Retinogram which is dominated by cones. His analysis of the electroretinogram is the most complete and best established to date. With his micro-electric technique Granit has fully confirmed the identity of the scotopic sensitivity curve and the visual purple absorption curve with its maximum at 500$\mu\mu$; and has shown that the photopic curve corresponds with the photopic luminosity curve with its maximum at 560$\mu\mu$. These he calls the scotopic and photopic dominator curves. These curves are further analyzed into

a definite number of curves which he calls the modulator curves. The dominator modulator theory shows that the scotopic and photopic curves are true luminosity curves, i. e. brightness differences.

The Appendix I on technical notes and Appendix II on electroretinography in man together with a large and complete reference table makes this a most valuable book for any one interested in neurologic or ophthalmic philology. The format is excellent and the numerous illustrations consisting chiefly of graphs and charts are comprehensive and accompanied by full explanatory notes. The book should be a must for anyone working in electrophysiology and the physiology of vision but in a general medical library it would probably find a very limited use.—*Capt. W. L. Berkley (MC) U. S. N.*

HANDBOOK OF ORTHOPEDIC SURGERY, by Alfred Rives Shands, Jr., B. A., M. D., *Medical Director of the Alfred I. duPont Institute of the Nemours Foundation, Wilmington, Del.; Visiting Professor of Orthopedic Surgery, University of Pennsylvania School of Medicine, Philadelphia, Pa.*, in collaboration with Richard Beverly Raney, B. A., M. D., *Associate in Orthopedic Surgery, Duke University School of Medicine, Durham, N. C.; Lecturer in Orthopedic Surgery, University of North Carolina School of Medicine, Chapel Hill, N. C.* 3d edition. 574 pages, illustrated. The C. V. Mosby Co., St. Louis, Mo., publishers, 1948. Price $6.

Dr. Shands first introduced this handbook in 1937 and since that time it has become a standard reference in undergraduate orthopedic instruction. It is also widely used by the pediatrician and the general practictioner, which testified to its suitability. In this most recent revision, Dr. Shands and his co-author, Dr. Raney, have added some new material which has evolved from experiences gained in the recent war. The original form and most of the original illustrations are retained.

The book systematically covers the general field of orthopedics, including basic considerations of bone and joint affections, proceeding through the congenital deformities to the affections of growing bone, and of adult bone. The arthritides and neuromuscular disabilities are adequately covered. Several chapters give consideration to the special regions of the musculo-skeletal systems. A brief summation of fractures is given. It is not the intention of this handbook to detail treatment, but merely to call attention to the deformities which may occur from trauma.

This book will continue to serve as an excellent outline for teaching medical undergraduates, nurses, physical therapists, and hospital corpsmen. The excellent bibliography makes it a valuable source book for the orthopedic resident and researcher.—*Commander C. R. Carr (MC) U. S. N.*

PRACTICE OF ALLERGY, by Warren T. Vaughan, M. D., *Richmond Va.* Revised by J. Harvey Black, M. D., *Dallas, Tex.* 2d edition. 1,132 pages, illustrated. The C. V. Mosby Co., St. Louis, Mo., publishers, 1948. Price $15.

This detailed second edition of a well-known classic in the field of allergy appears 9 years after the original edition. The death of the original author might have terminated the usefulness of what started as an outstanding text in this field, but fortunately Dr. Black has carried on, adding new knowledge and removing that which is no longer acceptable.

To the student new to this sphere, this volume offers what is too often missing in current texts—a keenly appreciative and revealing historical chapter. To all but those who use a book merely for rapid reference, such an introduction serves a great but intangible purpose in orientation and appreciation. This is followed in natural sequence by detailed chapters on the theories of anaphylaxis, the relation of experimental anaphylaxis and clinical allergy, terminology, and functional pathology. The discussions on the nature of allergy are particularly illuminating. The next step is a consideration in several chapters of the factors of incidence,

environment, heredity, and the growth of the allergic child, followed by three extremely detailed chapters on physiology. The psychological aspects of allergy are well covered.

The authors then move nearer the individual patient with an excellent discussion of the approach to the patient and the history of the patient's illness which is so extremely important in allergic diagnosis. All conceivable means of testing are taken up in several chapters with a very fair consideration of the pros and cons, and of the opinions of others.

Of use to those new to this field are detailed instructions relative to needles, syringes, extracts, and other material.

The rather controversial problems of food allergies are well presented and the approaches of any one authority are not unduly stressed.

Pollens, Polinosis, and other inhalant allergies are introduced by chapters on the development and distribution of pollens, botanical aspects, and surveying methods. There are many good and extremely helpful illustrations of various plants of allergic significance. There are several chapters on various means of treatment, reactions, and inhalants other than pollen.

Allergies related to bacteria, fungi, and animal products are considered with the same detailed approach. Anaphylactic shock, drug allergy, contact allergy, and physical allergy are well outlined. The chapter on the use of drugs in the treatment of allergy is extensive and includes the antihistaminics.

The last part of the book is devoted to the various allergic diseases in all their manifestations. A number of diseases which are of doubtful status are considered; however, there is no attempt to overwhelm the reader with arguments in favor of their allergic origin.

The book is well organized and clearly printed. One departure from many medical texts is a listing of references at the end of the book and the lack of reference numbers. While this makes the use of the references somewhat more difficult, it also makes for a more readable and nicer appearing volume.—*Commander R. C. Parker, Jr. (MC) U. S. N.*

ORAL AND DENTAL DISEASES, Aetiology, Histopathology, Clinical Features and Treatment, A Textbook for Dental Students and a Reference Book for Dental and Medical Practitioners, by Hubert H. Stones, M. D., M. D. S., F. D. S. R. C. S. (Eng.), *Professor of Dental Surgery and Director of Dental Education, University of Liverpool; Hon. Director, Liverpool Dental Hospital; Consultant, E. M. S. Maxillofacial Centre, Broad Green Hospital, Liverpool, formerly External Examiner in Dental Subjects, Universities of Bristol, Cambridge, Leeds and Manchester; External Expert in Dentistry, University of London; Hon. Dental Surgeon, University College Hospital Dental Department, London.* 896 pages; 926 illustrations. The Williams & Wilkins Co., Baltimore, Md., publishers, 1948. Price $18.

This book is a new publication by a British author. In this voluminous work an attempt has been made to cover etiology, histopathology, clinical features, and treatment of the diseases and injuries of the teeth, their investing tissues, the jaws, and oral mucosa.

The material presented in most chapters is brief. For example, treatment of jaw fractures covers five pages; treatment of impacted third molars covers two pages. Malocclusion of the teeth and maldevelopment of the jaws, usually written as a separate textbook, covers only 45 pages. However, an unusual feature is the comprehensive bibliography at the conclusion of each chapter, which should aid the reader to gather more material. The chapter on stains and discoloration of the teeth is well presented.

The format is good for this type of book. The illustrations are well done, especially those in color.

The author states this book is a textbook for dental students. Considering the brief and incomplete information in chapters on etiology of dental caries,

traumatic maxillo-facial injuries, and histopathology and clinical features of dental caries, its use is not recommended in American dental schools.—*Commander A. Bartelle (DC) U. S. N.*

HANDBOOK OF DENTAL PRACTICE, edited by Louis I. Grossman, D. D. S., Dr. Med. Dent., with 18 authors. 417 pages, illustrated. J. B. Lippincott, Philadelphia, Pa., publishers, 1948. Price $12.

This book is intended to serve as a daily guide in dental practice and for handy reference purposes. Concise description of technics is featured in a number of the chapters, while others are devoted principally to discussions of specialty practice problems or to the classification and treatment of various types of cases. The book is well printed and arranged and is divided into two parts, the first of which includes sections on anesthesia, caries prevention and control, oral diagnosis, and the dental practice specialties except those having to do with restorative treatment. The second part covers dental operative and prosthetic procedures, with a chapter on pedodontics. The illustrations, all in black and white, are generally good. Considering that the 18 authors were allowed only about 22 pages each, the book is not recommended as a substitute for standard texts. A surprising amount of very useful special knowledge, nevertheless, is provided by some of the contributions, notably Dr. J. R. Cameron's tersely informative chapter on exodontics and Dr. V. R. Trapozzano's chapters on partial and complete artificial dentures.—*Capt. H. R. Delaney (DC) U. S. N.*

HEADACHE AND OTHER HEAD PAIN, by Harold G. Wolff, M. D., *Professor of Medicine (Neurology) and Associate Professor of Psychiatry, Cornell University Medical College; Attending Physician, New York Hospital, New York.* 642 pages, illustrated. Oxford University Press, New York, N. Y., publishers, 1948. Price $12.

The symptom of headache is the most common complaint that is presented to the physician. It is frequently one of the most distressing of human discomforts but, in the past, little has been known of its nature and pathogenesis. In the past 15 years, Dr. Wolff and his fellow workers have intensely studied the cerebral circulation and the other pain-sensitive structures within the cranial vault. This text is an excellent summary of Dr. Wolff's work and his observations on the work of other investigators. The close observation of clinical symptomatology is noted throughout the text. The major criticism of the text is that it supposedly covers headache and other head pain, however, there is less than one page allotted to the "psychogenic" headache, and after following patients with all of the suggested diagnostic and therapeutic aids as outlined, one is definitely hesitant to say that "psychogenic" headache is found only in a small percentage of patients.

In a little over 20 chapters, Dr. Wolff covers the problem of the organic approach to headache very well. This book is an excellent reference on headache.—*Lt. W. H. Boswell (MC) U. S. N.*

NEUROSURGICAL PATHOLOGY, by I. Mark Scheinker, M. D., *Assistant Professor of Neuropathology and Assistant Professor of Medicine (Neurology) University of Cincinnati, College of Medicine; Neuropathologist and Attending Neurologist, Cincinnati General Hospital.* 370 pages, illustrated. Charles C Thomas, Springfield, Ill., publishers, 1948. Price $8.75.

This second book of Dr. Scheinker's series of three on neuropathology deals in considerable detail with injuries and tumors of the central nervous system. Other short chapters on cerebral swelling, cerebral abscesses and hydrocephalus are included.

The chapters on central nervous system injuries occupy more than a third of the volume. It deals in a repetitious manner with the affects of injury on the brain as a whole rather than locally. The evolutional principle of structural lesions and the extensive interaction between vascular supply and structural

alterations are two concepts which are emphasized. Skull fracture and superficial brain lacerations are not considered.

In the chapter on tumors, which occupies approximately one-half the book, Dr. Scheinker says that his aim is to present a simplified tumor classification based exclusively upon the use of preparations stained with hematoxylin and eosin. He states this simplified scheme makes it possible for every neurosurgeon to assume full responsibility for a correct post-mortem laboratory examination, without specialized laboratory assistance and without special training in neuropathology. Although he states that rare tumors are to be omitted, the discussion of subependymoma occupies twice as much space as the discussion of ependymoma. Similarly diffuse cerebral glioblastosis is discussed in greater detail than any other heading under gliomas. In the discussion of individual tumors insufficient diagnostic criteria are given and some are given undue emphasis. For instance it is stated that the most outstanding characteristic of glioblastoma multiforme is the pseudopalisade formation due to necrosis; and the characteristic tendency of the astroblasts to arrange themselves about blood vessels in typical radiating fashion is often sufficient to warrant diagnosis of astroblastoma from a hematoxylin and eosin section. It is felt that Dr. Scheinker has fallen short of his mark.

The chapters on cerebral swelling, cerebral abscess, and hydrocephalus are adequate. The book is profusely illustrated. Some of the illustrations are inferior in quality, some of insufficient magnification to illustrate the point being made and some are repetitious. The references and index suffice.

—*Lt. B. K. Black (MC) U. S. N.*

## BOOKS RECEIVED

Receipt of the following books is acknowledged. As far as practicable, these will be reviewed at a later date.

POLLEN-SLIDES STUDIES, by Grafton Tyler Brown, M. D. *Instructor in Clinical Medicine, Georgetown University School of Medicine, Consultant on Allergy, United States Public Health Service, Head, Division on Allergy, Doctors Hospital, Washington, D. C.;* with a foreward by Wallace M. Yater, M. D., M. S. (In Medicine), F. A. C. P. *Director, Yater Clinic, Civilian Consultant Army Institute of Pathology and Walter Reed General Hospital, formerly Professor of Medicine, Georgetown University School of Medicine, Washington, D. C.* 122 pages with 98 figures. Charles C Thomas, Springfield, Ill., publishers, 1949. Price $6.

INTRODUCTION TO PARASITOLOGY With Special Reference to the Parasites of Man, by Asa C. Chandler, M. S., Ph. D., *Professor of Biology, Rice Institute, Houston, Texas; former Officer-in-Charge, Hookworm Research Laboratory, School of Tropical Medicine and Hygiene, Calcutta, India.* 8th edition. 756 pages; illustrated. John Wiley & Sons, Inc., New York, N. Y., publishers, 1949. Price $6.

THE WORLD AS I SEE IT, by Albert Einstein. Translated by Alan Harris. 112 pages. Philosophical Library, New York, N. Y., publishers, 1949. Price $2.75.

THE MAGIC CLOAK, A Contribution to the Psychology of Authoritarianism, by James Clark Moloney, M. D. 345 pages, with illustrations by Erle Loran. The Montrose Press, Wakefield, Mass., publishers, 1949. Price $5.

NURSING IN CLINICAL MEDICINE, by Julius Jensen, Ph. D. (in Medicine), M. R. C. S. (Eng.)., L. R. C. P. (Lond.), *Assistant Professor in Medicine, Washington University, St. Louis; Member of Staff, St. Luke's Hospital, St. Louis; Diplomate of American Board of Internal Medicine (Cardiovascular Disease)*; and Deborah Maclurg Jensen, M. A., B. Sc., R. N., *Lecturer in Nursing Education, Washington University, St. Louis; Visiting Instructor at St. Louis City and St. Lukes Hospital, St. Louis; formerly Social Service Consultant, Visiting Nurse Association, St. Louis.* 3d edition. 791 pages. The Macmillan Co., New York, N. Y., publishers, 1949. Price $4.

ESSENTIALS OF GYNECOLOGY, by Leo Brady, M. D., F. A. C. S. *Assistant Professor of Gynecology, Johns Hopkins University; Assistant Professor of Gynecology, University of Maryland; Assistant Attending Gynecologist, Johns Hopkins Hospital; Consulting Gynecologist, Church Home and Infirmary, Hospital for the Women of Maryland, St. Joseph's, The Union Memorial and University Hospitals;* Ethna Louise Kurtz, R. N., *formerly, Head Nurse, Brady Urological Institute, Johns Hopkins Hospital; formerly Supervisor, Gynecological Operating Room, Johns Hopkins Hospital;* and Eileen McLaughlin, B. S., R. N., *Instructor and Supervisor, Gynecological Nursing, Johns Hopkins Hospital.* 2d edition. 256 pages, illustrated. The Macmillan Co., New York, N. Y., publishers, 1949. Price $3.

MARIHUANA IN LATIN AMERICA, The Threat It Constitutes, by Pablo Osvaldo Wolff, M. D., Ph. D., M. A., Buenos Aires, Argentina; *Member of Expert Committee on Habit Forming Drugs of the World Health Organization, sponsored by Washington Institute of Medicine.* 56 pages. The Linacre Press, Inc., Washington, D. C., publishers, 1949. Price $1.50.

PERIODONTIA, A Study of the Histology, Physiology, and Pathology of the Periodontium and the Treatment of Its Diseases, by Henry M. Goldman, D. M. D., *Chief of Stomatology and Head of the Dental Department, Beth Israel Hospital; Periodontist, Massachusetts General Hospital, Boston, Mass.; Consultant, Army Institute of Pathology, Washington, D. C.* 2d edition. 611 pages; with 488 illustrations including 18 in color. The C. V. Mosby Co., St. Louis, Mo., publishers, 1949. Price $12.50.

ATLAS OF OBSTETRIC TECHNIC, by Paul Titus, M. D., *Obstetrician-Gynecologist to the St. Margaret Memorial Hospital, Pittsburgh; Secretary, American Board of Obstetrics and Gynecology.* Illustrations by E. M. Shackelford, *formerly Medical Illustrator, John C. Oliver Memorial Research Foundation, St. Margaret Memorial Hospital, Pittsburgh.* 2d edition. 197 pages; illustrated. The C. V. Mosby Co., St. Louis, Mo., publishers, 1949. Price $7.50.

A DESCRIPTIVE ATLAS OF RADIOGRAPHS, An Aid to Modern Clinical Methods, by A. P. Bertwistle, M. C., Ch. B., F. R. C. S. Ed. 7th edition, revised and enlarged. 622 pages, with 980 illustrations. The C. V. Mosby Co., St. Louis, Mo., publishers, 1949. Price $15.

ISOTOPIC TRACERS AND NUCLEAR RADIATIONS, With Applications to Biology and Medicine, by William E. Siri, with contributions by Ellsworth C. Dougherty, Cornelius A. Tobias, James S. Robertson, Rayburn W. Dunn, and Patricia P. Weymouth, *Division of Medical Physics, Department of Physics, and Radiation Laboratory, University of California.* 653 pages; 136 illustrations. The McGraw-Hill Book Co., New York, N. Y., publishers, 1949. Price $12.50.

A PRACTICE OF ORTHOPAEDIC SURGERY, by T. P. McMurray, C. B. E., M. B., M. Ch., F. R. C. S. (Edin.). *Professor of Orthopaedic Surgery, Liverpool University, Honorary Orthopaedic Surgeon, David Lewis Northern Hospital, Director of Orthopaedics, Royal Liverpool Children's Hospital, Consulting Orthopaedic Surgeon, Lancashire County Council, Visiting Orthopaedic Surgeon, Alder Hey Children's Hospital, Liverpool, Consulting Surgeon to the Ministry of Pensions Hospital, Regional Orthopaedic Consultant, Ministry of Health, etc.* 3d edition. 444 pages; illustrated. The Williams & Wilkins Co., Baltimore, Md., publishers, 1949. Price $8.

ILLUSTRATIONS OF SURGICAL TREATMENT, Instruments and Appliances, by Eric L. Farquharson, M. D., F. R. C. S. (Ed.), F. R. C. S. (Eng.), *Assistant Surgeon, Royal Infirmary, Edinburgh; member of Clinical Teaching Staff, University of Edinburgh; Surgeon, Berwick-on-Tweed Infirmary; Assistant Surgeon, Kirkcaldy Hospital.* With a foreword by the late Sir John Fraser, Bt., K. C. V. O., M. C., M. D., Ch. M., F. R. C. S. (Ed.), *formerly Regius Professor of Clinical Surgery, University of Edinburgh.* 3d edition. 391 pages; illustrated. The Williams & Wilkins Co., Baltimore, Md., publishers, 1949. Price $7.

INJURIES OF THE BRAIN AND SPINAL CORD AND THEIR COVERINGS, Neuro-Psychiatric, Surgical, and Medico-Legal Aspects. Edited by Samuel Brock, *New York University,* with 28 contributors. 3d edition. 783 pages; illustrated. The Williams & Wilkins Co., Baltimore, Md., publishers, 1949. Price $10.

A COMPANION IN SURGICAL STUDIES, by Ian Aird, Ch. M., F. R. C. S., *Professor of Surgery in the University of London, Director of the Surgical Unit, Postgraduate Medical School of London.* 1060 pages. The Williams & Wilkins Co., Baltimore, Md., publishers, 1949. Price $15.

PHOTORADIOGRAPHY IN SEARCH OF TUBERCULOSIS, by David Zachs, M. D., *Chief of Clinics, Massachusetts Department of Public Health.* 297 pages; illustrated. The Williams & Wilkins Co., Baltimore, Md., publishers, 1949. Price $5.

STREPTOMYCIN, NATURE AND PRACTICAL APPLICATIONS, edited by Selman A. Waksman, Ph. D., *New Jersey Agricultural Experiment Station, Rutgers University,* with 58 contributors. 618 pages; illustrated. The Williams & Wilkins Co., Baltimore, Md., publishers, 1949. Price $10.

STEDMAN'S MEDICAL DICTIONARY, edited by Norman Burke Taylor, M. D., F. R. S. C., F. R. C. S. (Edin.), F. R. C. P. (Can.), M. R. C. S. (Lon.), *University of Western Ontario and formerly of the University of Toronto;* in collaboration with Allen Ellsworth Taylor, D. S. O., M. A. 17th revised edition with etymologic and orthographic rules. 1361 pages; illustrated. The Williams & Wilkins Co., Baltimore, Md., publishers, 1949. Price $8.50 with thumb index; $8 without thumb index.

TREATMENT IN PROCTOLOGY, by Robert Turell, B. S., M. D., *Attending Proctologist, Hillside Hospital; Adjunct Surgeon in Proctology, Montefiore Hospital; Adjunct Surgeon in Proctology, Beth Israel Hospital; Senior Clinical Assistant, Rectal Clinic, Mount Sinai Hospital, New York;* with a chapter on psychosomatic problems by Louis Linn, M. D. 248 pages; illustrated. The Williams & Wilkins Co., Baltimore, Md., publishers, 1949. Price $7.

THE ORIGIN OF MEDICAL TERMS, by Henry Alan Skinner, *M. B., F. R. C. S. (C). Professor of Anatomy, University of Western Ontario.* 379 pages. The Williams & Wilkins Co., Baltimore, Md., publishers, 1949. Price $7.

BLOOD AND PLASMA TRANSFUSIONS, by Max M. Strumia, M. D., Sc. D. (Med.), *Associate Professor of Pathology, Graduate School of Medicine, University of Pennsylvania; Director, Laboratory of Clinical Pathology, and of the John S. Sharpe Research Foundation, Bryn Mawr Hospital; Member, Subcommittee on Blood Substitutes of the National Research Council, 1940–1945;* and John J. McGraw, Jr., M. D., *Instructor in Pathology, Graduate School of Medicine, University of Pennsylvania; Assistant Attending Pathologist, Bryn Mawr Hospital; formerly, Commanding Officer of the Blood Bank for the Mediterranean Theater of Operations, Chief of the Blood Research Division of the Army Medical School, and Special Representative to the Surgeon General on Blood and Plasma Transfusion.* 497 pages; 124 illustrations. F. A. Davis Co., Philadelphia, Pa., publishers, 1949. Price $7.50.

FUNDAMENTALS OF PHARMACY, Theoretical and Practical, by Walter H. Blome, Ph. C., M. S., M. A., *Emeritus Professor of Pharmacy, Wayne University, College of Pharmacy, Detroit, Mich.,* and Charles H. Stocking, Ph. C., M. S., *Dean, University of Michigan College of Pharmacy, Ann Arbor, Mich.* With contributions by Elmon L. Cataline, Ph. D.; Robert L. Jones, Ph. C., M. S.; and Edward C. Watts, B. S. 2d edition, revised. 312 pages; with 158 illustrations. Lea & Febiger, Philadelphia, Pa., publishers, 1949. Price $5.

SURGICAL MANAGEMENT OF VASCULAR DISEASES, by Gerald H. Pratt, M. D., F. A. C. S. *Associate Clinical Professor of Surgery, New York University, Chief of the Vascular Clinic and Associate Attending Surgeon, Saint Vincent's Hospital, City of New York; Attending Surgeon, St. Clare's Hospital, City of New York; Diplomate of American Board of Surgery; Commander, Medical Corps, United States Naval Reserve.* 496 pages; illustrated. Lea & Febiger, Philadelphia, Pa., publishers, 1949. Price $10.

A TEXTBOOK OF NEUROPATHOLOGY, with Clinical, Anatomical and Technical Supplements, by Ben W. Lichtenstein, B. S., M. S., M. D., *Associate Professor of Neurology, the University of Illinois College of Medicine; State Neuropathologist, Illinois Neuropsychiatric Institute; Attending Neurologist, Cook County Hospital; Professor of Neurology, the Cook County Graduate School of Medicine; Attending Neuropsychiatrist, Mount Sinai Hospital, Chicago.* 474 pages with 282 figures. W. B. Saunders Co., Philadelphia, Pa., publishers, 1949. Price $9.50.

CLINICAL BIOCHEMISTRY, by Abraham Cantarow, M. D., *Professor of Biochemistry, Jefferson Medical College,* and Max Trumper, Ph. D., *Commander, H(S), USNR. Lecturer in Clinical Biochemistry and Basic Science Coordinator, Naval Medical School, National Naval Medical Center, Bethesda, Md.* 4th edition. 642 pages with 38 figures. W. B. Saunders Co., Philadelphia, Pa., publishers, 1949. Price $8.

○